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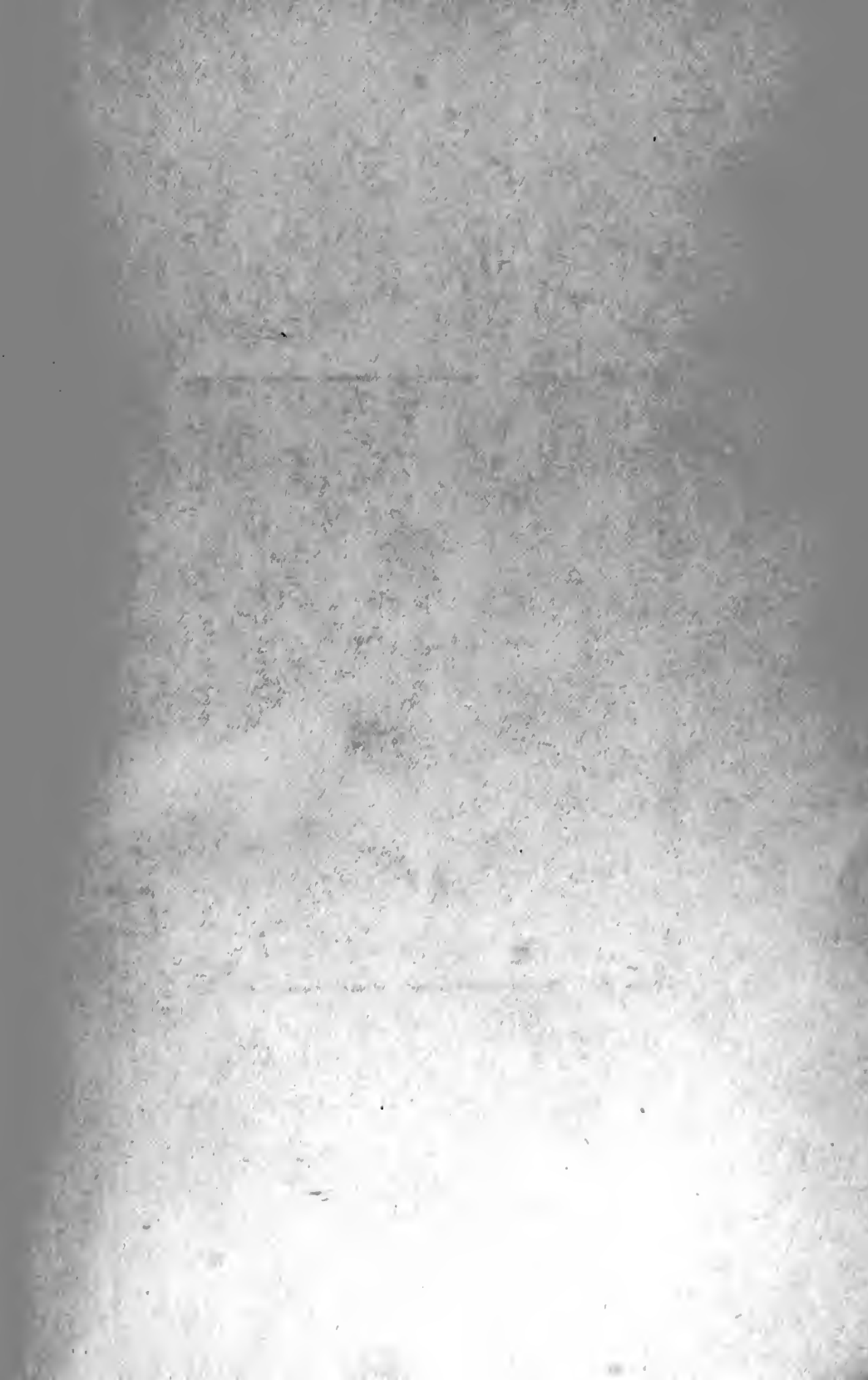
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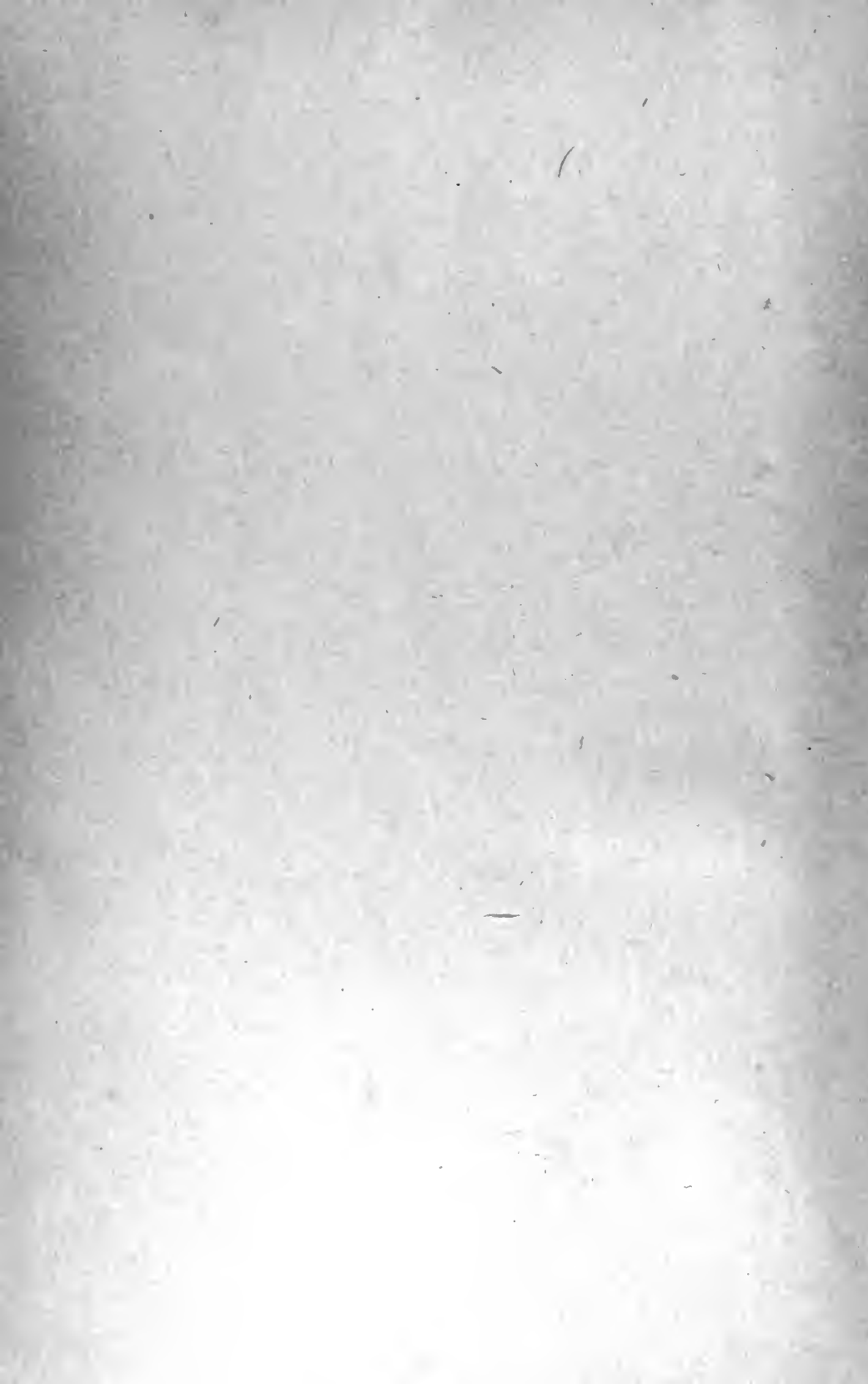
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THE DISEASES OF INFANTS AND CHILDREN

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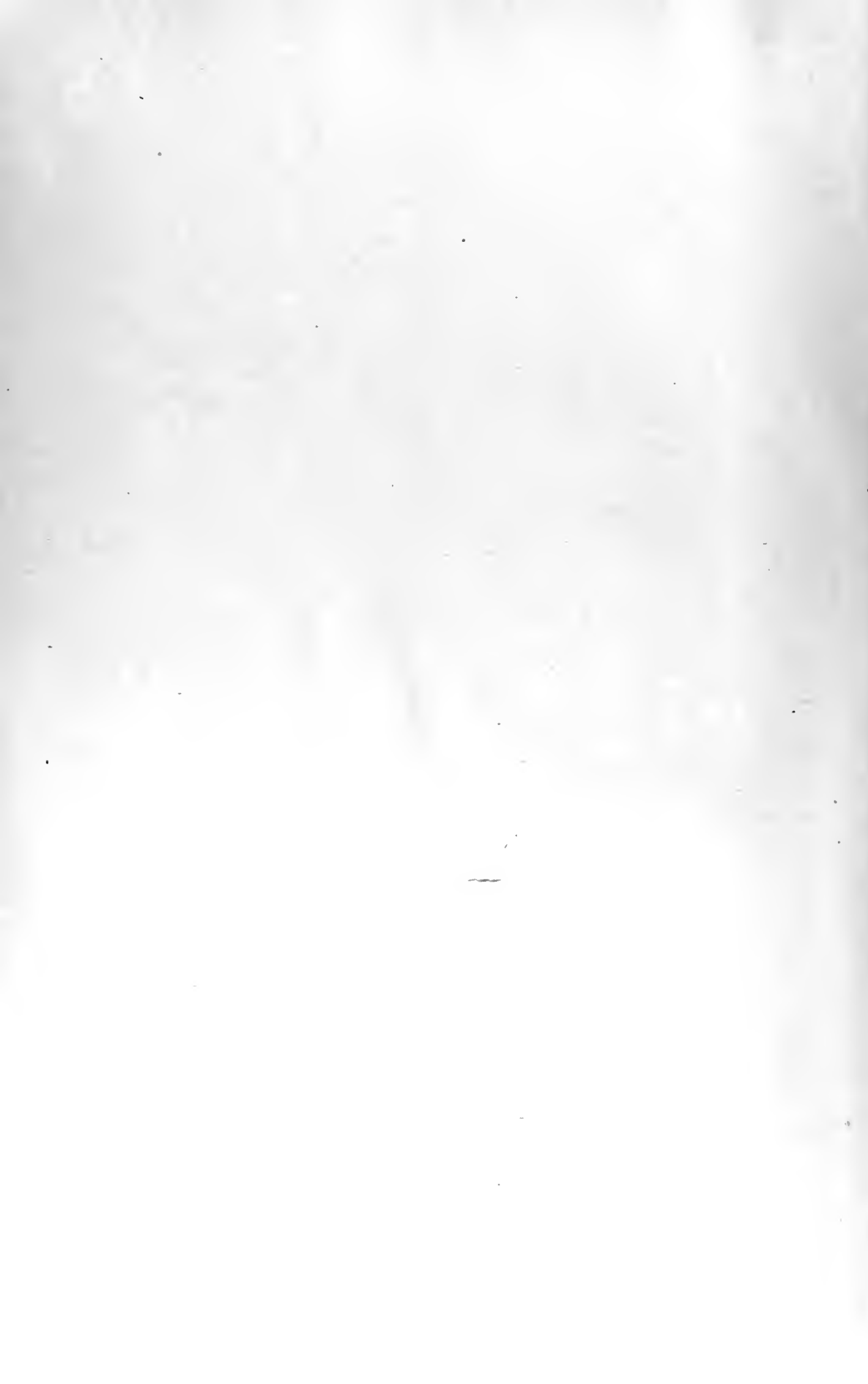
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v. 1

DEDICATED TO THE MEMORY OF
JAMES TYSON, M.D., LL.D.
PHYSICIAN, CLINICIAN, GENTLEMAN.
MY ONE-TIME TEACHER AND
ALWAYS MY TRUSTED ADVISER AND FRIEND



PREFACE

It has been the effort of the author in the following pages to present a review of the subject of medical pediatrics, as complete as seemed desirable without attempting to make it encyclopedic. Inclusion is made of such subjects in surgery and the special branches with the recognition of which physicians treating the diseases of children should be more or less familiar. While endeavoring to embody the results of his own experience through many years of contact with disease in children, he has also made free use of the numerous excellent text-books on the subject, including the valuable contributions by American authors, and of home and foreign pediatric journal-literature. To all these authors he would here acknowledge his indebtedness.

In the course of his own reading he has found quotations from medical authorities of much impaired service unless accompanied by reference to the places of publication, thus rendering possible the consulting of the originals. With the feeling that others may share this sentiment, he has in footnote form given the references to literature whenever such quotations are used, believing that in this way the value of the book to many readers would be increased, while the footnote method interferes in no way with its usefulness to those others who are not interested in this line of research.

Temperature-charts and photographic and other illustrations have been reproduced freely, generally accompanied by brief synopses of the histories of the cases, without which their value would be much lessened. These are original or unpublished except in the instances where none such were obtainable, or where superior ones were found in the publications of other writers. Acknowledgment has naturally been made in every case.

Throughout the text-book the metric and the English systems of measurements have been used together, putting in parentheses in the terms of one the equivalents in the other. The statistics quoted from any author have been given in the system employed by him, and the corresponding figures in the other then placed in parentheses. The equivalent values are largely those found in the tables of the United States Pharmacopœia. Ounces are respectively avoirdupois or liquid measure, except in designating the doses of solid medicaments, when Troy ounces are used. Fractional amounts in grains, drams, cubic millimeters, cubic centimeters, and grams are omitted unless the quan-

tities are small. Grams are assumed to be the equivalent of cubic centimeters, ignoring the specific gravity of many liquids, where the figures as given would not be absolutely correct. In the parentheses the abbreviations designating grams and cubic centimeters are omitted, the sense of the text making them unnecessary. An exception to the employment of both systems of measurement will be found in discussing the preparation of food in the artificial feeding of infants. Here only English measures are given, since the preparation must be made in conjunction with the graduated nursing-bottles and the liquid measures in common household use.

Numerous cross-references will be found throughout the work, thus calling attention to discussions of the subjects on other pages, which would otherwise be overlooked unless the index were consulted. Although every effort has been made to avoid inaccuracy of statements, and particularly of references, the author must expect to share the experience of others, that these will creep in to some extent.

The author has waived his own preferences in the matter, yielding to the desire of the publishers for uniformity in the system of spelling and of punctuation adopted throughout the numerous works upon medical subjects published by them.

1810 SPRUCE STREET,
PHILADELPHIA, PA.,
September, 1919.

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THE DISEASES OF CHILDREN

DIVISION I

GENERAL SUBJECTS

CHAPTER I

ANATOMY AND PHYSIOLOGY

THE DIVISIONS OF LIFE

For the purpose of study human life is usually sub-divided into several stages. These are, of course, somewhat arbitrary divisions, and vary considerably with different writers. As convenient an arrangement as any is that which separates life into *Intra-uterine*, or fetal life; *Infancy*, including the *New-Born* as a sub-division; *Childhood*, early and later; *Youth* or adolescence, with *Puberty* as the initial period of this stage; *Adult* life, and its sub-division, *Old Age*.

Intra-uterine Life.—Although apparently closely resembling a new-born child, the fetus is in reality so different that it is not capable of independent existence. Many of its organs, though perfect, are not yet active. Circulation is not in the complete state which it will later assume. Respiration and digestion are entirely in abeyance, nutriment and oxygen being obtained through the maternal blood.

Being so closely dependent upon the mother for its life, it follows that the condition of her health and her methods of living cannot fail to exert a great influence upon the health of the fetus, and, indeed, upon the whole later existence of the child.

The fetus may also directly inherit a feeble constitution from a delicate father or mother, irrespective of the production of debility by the preventable causes mentioned. Actual disease may occasionally be transmitted from mother to fetus. This is true of many of the infectious disorders, such as measles, whooping cough, typhoid fever, malaria, syphilis, tuberculosis and the like. The tendency to rheumatism or to gout is directly transmitted, as are certain nervous disorders, although the latter are oftener the result of other nervous conditions in the parents, or of imperfect hygiene of the nervous system of the mother while pregnant.

There are, besides, a variety of accidents which may happen to the fetus *in utero*, such as fractures, dislocations, amputations and the like. Various diseases may occur, or anomalies of development arise, producing monsters or lesser degrees of malformations, the reason for the occurrence of which is little understood so far as any preventive measures are concerned. Other characteristics and disorders of this period are fully

treated of in works upon obstetrics and diseases of the embryo and fetus. The condition of development seen in infants the subject of *premature birth* will be considered when discussing that topic (p. 252).

The New Born.—The first period of infancy is that in which the child is designated as *New Born*. This term is applied not only to the time immediately after birth, but to a slightly longer period, regarding the exact duration of which there is some variance of opinion. Bouchut¹ and Rilliet and Barthez,² for instance, limited it to the first few days of life; while Henoch³ extends it to the age of 4 to 6 weeks, and Parrot to 3 months.⁴ It seems best to apply the title to the first 2 or, at longest, 3 weeks, since in this period no very distinct alterations show themselves during the process of development. The change from intra-uterine to extra-uterine life is so great that the new born are very subject to disturbances of health, especially to those accidents and disorders caused by birth or developing shortly after it. There are also a number of pathological conditions seen at birth which developed during fetal life (p. 251).

Infancy.—This taken as a whole is a division of life to which various arbitrary and rather confusing limitations have been set. A classification common especially among French and German writers makes infancy (*première enfance*; *Säuglingsalter*) close with the age of 7 or 8 months, the beginning of dentition, or with the age of 1 year. Childhood (*seconde enfance*; *Kindersalter*) now begins—the first portion of it up to the age of about 36 months being called the *period of the first dentition*—and terminates with the age of 6 years. Youth (*Knabensalter*) then commences with 6 years, and forms the period of the second dentition, lasting until puberty. Popularly, however, in English-speaking countries infancy is generally considered as lasting until the age of 2 years and this seems the most convenient and best definition, since during this period the important epoch of the first dentition is nearly closed, and features are exhibited especially in the earlier part of it which differ in many respects from those seen in the later years of child-life. Children in the first year may be called *nurslings*.

Development, both of the body and of the mind, during infancy is very rapid, except that for the first few months there is little discoverable gain in intellectual power. Infants, especially those of an early age, are extremely susceptible to external depressing influences, such as cold and fatigue, and often easily affected by drugs, and rapidly exhausted by disease. On the other hand, they exhibit remarkable recuperative power when recovery begins.

Childhood.—According to the classification adopted here the term childhood is best used to cover the period from the close of infancy to the development of puberty. It may conveniently be divided into *Early Childhood* from the age of 2 to that of 6 years, the latter marking the beginning of the second dentition, and *Later Childhood* (*Knabensalter*) from the age of 6 years to puberty.

During childhood growth continues with a rapidity which, though great, is less than in infancy. The mental and physical differences between the sexes become constantly more apparent. The nervous system is still much in evidence although to a less extent than in infancy. The

¹ *Maladies des nouveau-nés*, 1885, 1.

² *Sanné, Maladies des enfants*, 1884, I, 5.

³ *Vorlesungen über Kinderkrankheiten*, 1895, 21.

⁴ *Clinique des nouveau-nés*, 1877, 4.

incidence of diseases at different periods of infancy and childhood will be discussed later (p. 209).

Youth.—According to the generally accepted definition the term *Youth* or *Adolescence* is applied to the period beginning with puberty and terminating with the commencement of adult life; *i.e.*, 21 to 25 years. The exact time for the development of puberty varies not only with individuals but with race and climate. In general for temperate climates it may be placed at from 14 to 16 years for boys and from 12 to 15 years for girls. Common law places it at 14 years for males and 12 years for females. With the occurrence of puberty the sexual functions are established in both sexes, the genital organs increase in size, and the growth of hair begins upon the pubis and later in the axillæ. In the female the breasts enlarge and become rounded by a deposit of fat. In the male the voice changes and hair begins to grow upon the face. The psychic characteristics belonging to each sex now become accentuated. The attributes and diseases most peculiar to youth do not, of course, come under special consideration in the study of pediatrics.

THE NEW BORN

The infant at birth is more or less covered by a white, waxy substance, the *vernix caseosa*, which protected it during fetal life. This is especially abundant on the flexor surfaces, the back and in the folds of the body, although some children have almost none of it upon them. It is composed of the thickened secretions of the sebaceous glands and of scales of epidermis. When it has been removed by washing the skin is found to be

thin, smooth, delicate and of a deep-reddish color. The lanugo characteristic of fetal life has generally largely disappeared in fully developed new-born children. Many infants, however, still show a fine soft down.

The child's flesh should be plump and firm from a good development of subcutaneous fat. The head is proportionately very large and often rather thickly covered with long hair. The eyes are an indeterminate blue, and are usually kept shut or but half open. The face is expressionless. The chest is small and narrow and the abdomen large and prominent. The arms are short and are held most of the time flexed and pressed against the body in the position maintained in the uterus. The hands are generally closed, but will grasp firmly any object placed in them. The nails are well developed, and project beyond the tips of the fingers and toes. The legs are comparatively small and short, and apparently curved with an outward bow (Fig. 1). They are much of the time held flexed at the knees and drawn up to the abdomen as in the fetal



FIG. 1.—BABY A FEW WEEKS OLD, SHOWING THE NATURAL CURVE OF THE LEGS WITH THE BENDING IN OF THE SOLES. (From a photograph.)

position. Intellectual activity is practically absent and the power of the special senses largely in abeyance.

DEVELOPMENT

A more detailed study is necessary of the characteristics of the new-born child and of its development as growth advances. These subjects will be considered in course.

Increase in Weight.—The weight of the normal healthy child at birth may be taken as from 7 to $7\frac{1}{2}$ lb. (3175 to 3402). The children of primiparæ are slightly lighter than those of multiparæ, the difference averaging about 5 oz. (142). Investigations by Dr. J. C. Gittings and myself upon 226 new-born infants gave an average initial birth-weight of 3455.79 grams (7.62 lb.). There is, however, a great range in the weight of the new born even within physiological limits. This is shown by the variation in the statistics, as seen in the following table:

TABLE 1.—AVERAGE WEIGHT AT BIRTH

Kézmásky ¹	73 cases	Average 3330 grams (7.34 lb.)
Ingerslev ²	3450 cases	Average 3334 grams (7.35 lb.)
Holt ³	1158 cases	Average 3330 grams (7.34 lb.)
Schäffer ⁴	94 cases	Average 3151 grams (6.95 lb.)
Camerer ⁵	119 cases	Average 3433 grams (7.57 lb.)
Peterson ⁶	1675 cases	Average 3527 grams (7.78 lb.)
Griffith and Gittings ⁷	226 cases	Average 3456 grams (7.62 lb.)
Fuhrmann ⁸	1000 cases	Average 3337 grams (7.36 lb.)

Male children are somewhat heavier than female, the difference being about 100 to 200 grams (3.5 to 7 oz.). In the observations of Gittings and myself 111 males averaged 3494.06 grams (7.70 lb.), and 115 females 3418.08 grams (7.54 lb.). Some of the statistics illustrating the differences between the sexes are seen in the following table:

TABLE 2.—BIRTH-WEIGHT OF MALE AND OF FEMALE INFANTS RESPECTIVELY

	Males	Females
Ingerslev ⁹	3381 grams (7.45 lb.)	3280 grams (7.23 lb.)
Gregory ¹⁰	3386 grams (7.46 lb.)	3331 grams (7.34 lb.)
Quetelet ¹¹	3200 grams (7.05 lb.)	2910 grams (6.41 lb.)
Altherr ¹²	3214 grams (7.08 lb.)	3077 grams (6.78 lb.)
Kézmásky ¹³	3383 grams (7.46 lb.)	3284 grams (7.21 lb.)
Holt ¹⁴	3400 grams (7.49 lb.)	3260 grams (7.24 lb.)
Peterson ¹⁵	3595 grams (7.92 lb.)	3455 grams (7.62 lb.)
Griffith and Gittings ¹⁶	3494 grams (7.70 lb.)	3418 grams (7.54 lb.)

¹ Arch. f. Gynäk., 1873, V, 547.

² Nord. Med. Ark., 1875, VII, No. 7, 8.

³ Dis. of Inf. and Childh., 1911, 16.

⁴ Arch. f. Gynäk., 1896, LIII, 616.

⁵ Jahrb. f. Kinderheilk., 1901, LIII, 413.

⁶ Upsala läkeref. förhandl., 1882, XVIII, 15.

⁷ Arch. of Ped., 1907, XXIV, 321.

⁸ Med. Klinik, 1907, III, 510.

⁹ Loc. cit.

¹⁰ Arch. f. Gynäk., 1871, 52.

¹¹ Sur l'homme et le développement, etc., 1836, II, 49.

Ref. Fleischman, Wiener Klinik, 1877, June and July.

¹² Ueber regelmässige Wägung der Neugeborenen. 1874.

¹³ Loc. cit.

¹⁴ Loc. cit.

¹⁵ Loc. cit.

¹⁶ Loc. cit.

Immediately after birth the loss of weight begins in nearly all cases. This depends upon the excretion of meconium and urine, the loss of the vernix caseosa, the excretions of the skin and lungs and the metabolic changes progressing in the tissues. The weight of meconium, urine and vernix ranges from 3 to 5 oz. (85 to 142) according to the observations of Townsend.¹ A review made by Fleischman² of the results obtained by a number of observers showed it to average 222 grams (7.83 oz.); i.e., about $\frac{1}{15}$ (6.66 per cent.) of the whole weight if we assume this as 3300 grams (7.28 lb.). Certain other observations, however, give a loss somewhat greater than this as physiological. In an examination of 226 infants made by Gittings and myself³ the total average loss equalled about 11 oz. (312), the relative loss being approximately $\frac{1}{11}$ (9 per cent.) of the initial weight. It is usually accepted that the heavier the full-term baby, the greater will be the absolute loss of weight; and it would appear from the studies of Longridge⁴ that the relative loss is also greater. Thus in 400 infants examined by him, the 7-lb. children lost $\frac{1}{14}$ of their body-weight, and the 5-lb. children only $\frac{1}{16}$. Pies's⁵ studies did not confirm this. The loss of weight is physiological, and usually cannot be entirely prevented. Observations carried out by Gittings and myself⁶ on 61 infants confirmed the results of Cramer⁷ and others, that by wet-nursing from the beginning until the mother's secretion is established, the initial loss of weight could be reduced very considerably, yet that this offered no great advantage. Keilman⁸ found that when artificial feeding was commenced immediately after birth, the infants, although losing somewhat less, regained the initial weight more slowly than did those who had been nourished solely at the mother's breast.

The diminution of weight continues until the 3d or 4th day and sometimes even longer, but cannot in the latter event be called physiological. The greatest loss takes place on the 1st day, from 3.5 to 4 per cent., and the next upon the 2d day, 2 to 2.6 per cent. of the birth-weight. The 3d day shows usually but little loss or the beginning of gain. The original weight is not regained before the 8th or 9th day, and often not until the 14th day. In a study upon 600 infants by Schulz,⁹ 288, or 48 percent., had regained their original weight in 10 days, illness of a number of the children preventing an earlier average time of regain.

Published statistics regarding the influence of sex are contradictory, some showing a greater loss in males, others in females. In our own¹⁰ studies upon 105 infants, the average loss in boys was somewhat greater and the regain of the initial weight slower in being accomplished. Townsend¹¹ noticed that children of primiparæ lose about $1\frac{1}{2}$ oz. (42) more than those of multiparæ.

The chart which follows¹² (Fig. 2.) shows the loss and regain of

¹ Boston Med. and Surg. Journ., 1887, CXVI, 157.

² Wiener Klinik, 1877, June-July.

³ *Loc. cit.*

⁴ Brit. Jour. Child. Dis., 1905, II, 403.

⁵ Monatsschr. f. Kinderh., Orig., 1910, IX, 514.

⁶ *Loc. cit.*

⁷ Münch. med. Woch., 1900, XLVII, 1585.

⁸ Jahrb. f. Kinderh., 1896, XLI, 312.

⁹ Inaug. Dissert. Greifswald, 1903. Ref. Arch. f. Kinderh., 1901, XXXIX, 207.

¹⁰ Griffith and Gittings, *loc. cit.*, 336.

¹¹ *Loc. cit.*

¹² New York Med. Journ., 1889, March 4.

weight in the first 10 days according to the observations of a number of investigators.

After gain in weight begins increase is rapid. Various estimations have been made of its rate, some of the more important of which are shown in graphic form in the illustration (Fig. 3).

Quetelet¹ assumed that growth continues at the same rate throughout the first year; hence his line in the chart is a *straight* one. Later investigations show that the rate of growth progressively decreases, this factor making the graphic plotting a *curved* line.

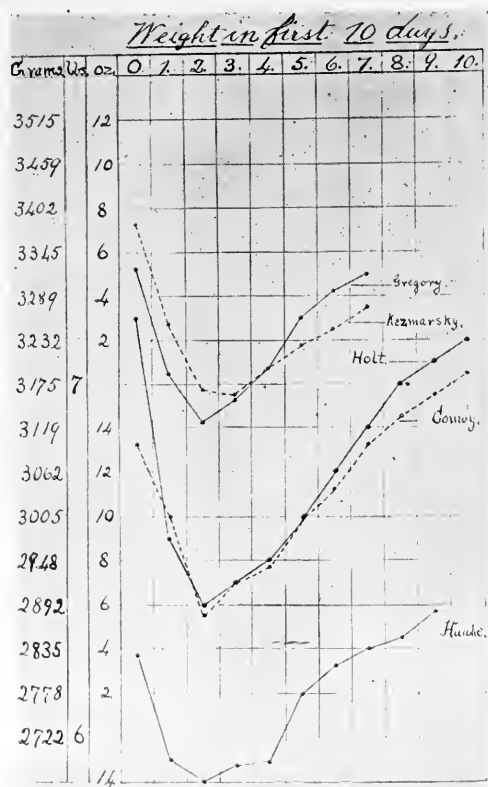


FIG. 2.—GRAPHIC CURVES SHOWING GAIN AND LOSS OF WEIGHT IN THE FIRST 10 DAYS OF LIFE.

According to different observers. (Griffith, *New York Med. Journ.*, 1899, March 4.)

It must be remembered that all tables of weight represent only the average rate of growth. A considerable variation in the individual child within physiological limits is possible.

The following figures give approximate weights of well-developed breast-fed children during the first year of life. They follow to a considerable extent the investigations of Camerer² and start with a birth-weight somewhat higher, viz., 3400 grams (7½ lb.), than that given by many investigators.

¹ Ref. Fleischmann, *loc. cit.*

² *Jahrb. f. Kinderh.*, 1893, XXXVI, 249.

TABLE 3.—SHOWING INCREASE IN WEIGHT DURING THE FIRST YEAR

Age	Weight	
Birth.....	7 lb. 8 oz.	(3402)
1 week.....	7 lb. 7½ oz.	(3388)
2 weeks.....	7 lb. 10½ oz.	(3473)
3 weeks.....	8 lb. 2 oz.	(3685)
1 month.....	8¾ lb.	(3969)
2 months.....	10¾ lb.	(4876)
3 months.....	12¼ lb.	(5557)
4 months.....	13¾ lb.	(6237)
5 months.....	15 lb.	(6804)
6 months.....	16¼ lb.	(7371)
7 months.....	17¼ lb.	(7824)
8 months.....	18¼ lb.	(8278)
9 months.....	18¾ lb.	(8505)
10 months.....	19¾ lb.	(8958)
11 months.....	20½ lb.	(9299)
1 year.....	21½ lb.	(9752)

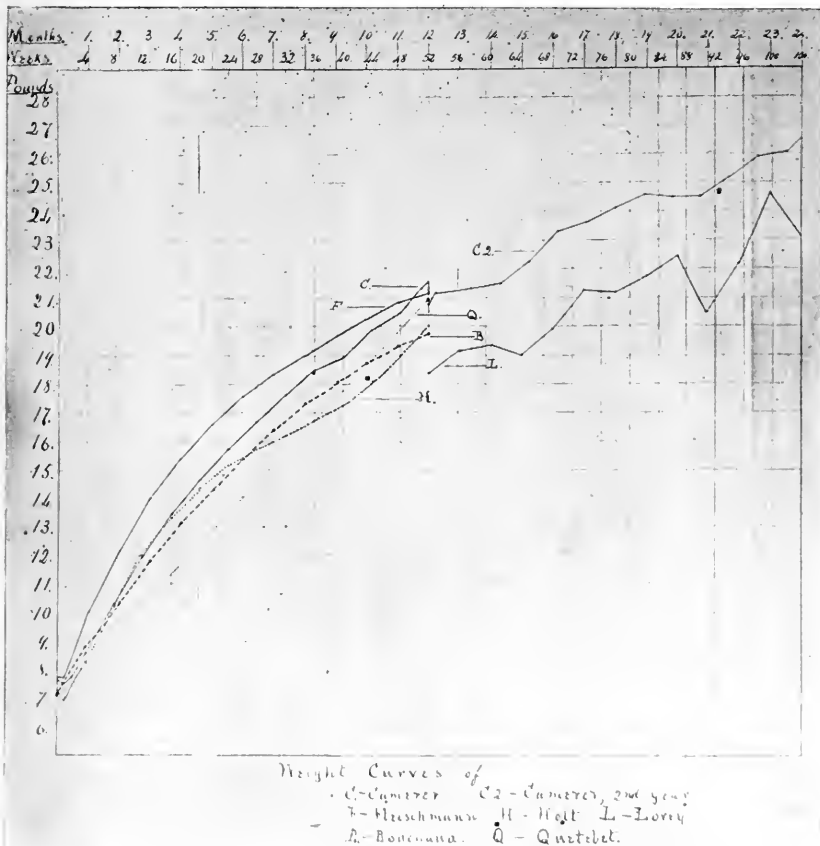


FIG. 3.—GRAPHIC CURVES SHOWING THE NORMAL GAIN IN WEIGHT DURING THE 1ST AND 2D YEARS.

Several observers. (Griffith, *New York Med. Journ.*, 1899, March 4.)

Analyzing these figures it will be noticed that in the last 3 weeks of the 1st month and during the 2d month the gain in weight is about 1 oz. (28) a day, 7 oz (198) a week; in the 3d and 4th months slightly

over $\frac{3}{4}$ of an oz. (21) a day, $5\frac{1}{2}$ oz. (156) a week; in the 5th and 6th months $\frac{2}{3}$ of an oz. (19) a day, $4\frac{2}{3}$ oz. (132) a week, and in the remainder of the 1st year about $\frac{1}{2}$ oz. (14) a day, $3\frac{3}{4}$ oz. (106) a week, or 1 lb. (454) a month; except that somewhere about the 9th month there is liable to be a temporary diminution in the rapidity of increase.

The initial average weight is doubled at the age of 5 months and trebled at the age of 1 year. Rauditz¹ has formulated a certain law according to which the increase in weight shall bear a certain definite mathematical relation to the age. The application of the rule is, however, not very practical.

The weight of males during the 1st year is on the average somewhat greater than that of females, amounting at the age of a year to a difference of $\frac{1}{2}$ up to even $1\frac{1}{4}$ lb. (227 to 567). Children fed artificially generally gain weight less rapidly than those at the breast, but this is by no means an invariable rule. This difference in weight depending on the food may persist during the first 3 or 4 years, but very often a healthy infant artificially fed and thriving on its diet, even although temporarily behind, will equal the breast-fed child in weight by the end of the 1st year.

There are but few extended observations made upon the weight of children from the 1st to the 6th year. Those of Camerer² appear reliable. The weights obtained by him are as follows:

TABLE 4.—INCREASE IN WEIGHT FROM THE SECOND TO THE FIFTH YEAR OF LIFE INCLUSIVE

(Camerer)

12 months.....	10,030 grams (22.11 lb.)
13 months.....	10,220 grams (22.53 lb.)
14 months.....	10,600 grams (23.36 lb.)
15 months.....	10,870 grams (23.96 lb.)
16 months.....	10,900 grams (24.03 lb.)
17 months.....	11,450 grams (25.24 lb.)
18 months.....	11,480 grams (25.30 lb.)
19 months.....	11,850 grams (26.12 lb.)
20 months.....	12,050 grams (26.56 lb.)
21 months.....	11,950 grams (26.34 lb.)
22 months.....	12,200 grams (26.80 lb.)
23 months.....	12,480 grams (27.51 lb.)
2 years.....	12,740 grams (28.08 lb.)
3 years.....	14,930 grams (32.91 lb.)
4 years.....	16,410 grams (36.16 lb.)
5 years.....	18,710 grams (41.25 lb.)

The studies of Perret and Planchon³ give figures about 100 grams (3.53 oz.) less than these. Those of Freeman⁴ upon 278 well cared for children in private practice gave results decidedly higher, as is to be expected. In general it may be assumed that the average healthy infant gains in its 2d year from 5 to 6 lb. (2268 to 2722); *i.e.*, $\frac{1}{2}$ lb. (227) a month or $\frac{1}{4}$ oz. (7) a day, the increase in the 1st half of the year being greater than in the 2d half. (See chart, p. 28.) In the 3d and 4th years the child gains about 5 lb. (2268) and in the 5th year about 4 lb. (1814). In tabular arrangement the figures read as follows:

¹ Prag. med. Wochenschr., 1892, Nos. 7 and 8.

² Jahrb. f. Kinderheilk., 1901, LIII, 418.

³ L'Obstetrique, 1904, IX, 193.

⁴ Amer. Jour. Dis. Child., 1914, VIII, 321.

TABLE 5.—GAIN IN WEIGHT FROM ONE TO FIVE YEARS

End of 1st year weighs	21½ lb. (9,752)	
End of 2d year weighs	27 lb. (12,247)	Gained 5½ lb. (2495)
End of 3d year weighs	32 lb. (14,515)	Gained 5 lb. (2268)
End of 4th year weighs	37 lb. (16,783)	Gained 5 lb. (2268)
End of 5th year weighs	41 lb. (18,597)	Gained 4 lb. (1814)

Girls continue to be from 1 to 1½ lb. (454 to 680) lighter than boys to the end of this period.

The following table shows the mean rate of increase for the two sexes according to the observations of Camerer:¹

TABLE 6.—GAIN IN WEIGHT OF BOYS AND GIRLS RESPECTIVELY
(Camerer)

	Boys	Girls
1st year.....	10,310 grams (22.73 lb.)	9,460 grams (20.86 lb.)
2d year.....	13,210 grams (29.12 lb.)	12,010 grams (26.48 lb.)
3d year.....	15,460 grams (34.08 lb.)	13,970 grams (30.78 lb.)
4th year.....	16,810 grams (37.06 lb.)	15,720 grams (34.66 lb.)
5th year.....	19,300 grams (42.59 lb.)	17,540 grams (38.67 lb.)

Estimating roughly the combined average weight of the sexes we find children gaining about 4 lb. (1814) in the 6th and 7th years, 4½ lb. (2041) in the 8th, 5 lb. (2268) in the 9th, 5½ to 6 lb. (2495 to 2722) in the 10th and 11th years, 7 to 8 lb. (3175 to 3629) in the 12th year, and then from 9 to 10 lb. (4082 to 4536) a year to the age of 16 years. In tabular form the approximate weight for the different years according to this rate of increase reads as follows:

TABLE 7.—WEIGHT FROM SIX TO SIXTEEN YEARS

6 years.....	45 lb. (20,412)
7 years.....	49 lb. (22,226)
8 years.....	53½ lb. (24,267)
9 years.....	58½ lb. (26,535)
10 years.....	64 lb. (29,030)
11 years.....	70 lb. (31,751)
12 years.....	78 lb. (35,380)
13 years.....	87 lb. (39,463)
14 years.....	96 lb. (43,549)
15 years.....	106 lb. (48,080)
16 years.....	116 lb. (52,617)

It will be seen that the child nearly doubles its weight at 1 year by the age of 5 years, and trebles it by 10 years.

The difference, however, which the sexes show in the rate of increase in weight is so decided in later childhood and in youth, that a separate consideration is necessary. The rate of growth continues about the same in each sex until about the age of 8 or 9 years, at which time the increase in girls is somewhat retarded, and boys advance decidedly beyond them. By 11 years, however, girls begin to gain in weight rapidly, and by 12 years decidedly surpass boys. This continues until the age of 15 or 16 years when boys again take and maintain the lead. The relative weights and rates of increase in boys and girls respectively during this period, according to the statistics of Bowditch,² Porter³

¹ *Loc. cit.*² 8th Ann. Rep. Mass. State Board of Health, 1877, 275.³ Transac. Acad. Science, St. Louis, VI, No. 12, 312.

and Peckham,¹ can be learned from the following table. The figures of Bowditch are based upon 24,595 observations upon public school-

children of Boston; those of Porter upon 30,817 children of St. Louis, and those of Peckham upon 5403 children of Milwaukee.

The figures given in this table are those for children of 5 years and over including clothing. Those for younger periods (p. 25) are without clothing. The average weight of the clothes, as estimated by Schmidt-Monard² is 6 per cent. to 7 per cent. of the weight of the body at the age of from 3 to 6 years, and 7 per cent. to 8 per cent. from 6 to 14 years. According to a series of personal estimations made on over 200 children, the average weight of the clothes in the 1st year is approximately $1\frac{3}{4}$ lb. (794); from 1 to 4 years, 2 lb. (907); and from 4 to 6 years, $2\frac{1}{4}$ to $2\frac{1}{2}$ lb. (102 to 113). The ratio of the weight of the clothing to the gross weight of the normal child dressed is approximately 16 per cent. in the first 3 months, but after this during the first 2 years approximately 8 per cent. to 10 per cent.; and from 2 to 5 years 6 per cent. Bowditch³ estimates the weight of the clothes at 5 years for boys as 2.8 lb. (1270); equalling 6.45 per cent. of the total gross weight for boys, and for girls as 2.84 lb. (1288) or 6.79 per cent. of the total weight for that sex; at 8 years 4 lb. (1814) or 7.23 per cent. for boys and 3.5 lb. (1587) or 6.54 per cent. for girls; at 11 years 6.7 lb. (3039) or 9.88 per cent. for boys, and 4.9 lb. (2223) or 6.88 per cent. for girls; at 14 years 8.1

TABLE 8.—WEIGHT OF BOYS AND GIRLS FROM FIVE TO EIGHTEEN YEARS

Years	Bowditch				Porter				Peckham			
	Boys		Girls		Boys		Girls		Boys		Girls	
	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms	Pounds	Kilograms
5	41.09	18.64	39.66	17.99	41.09	18.64	40.03	18.16
6	45.17	20.49	43.28	19.63	43.54	19.75	41.74	18.93	44.81	20.33	43.12	19.56
7	49.07	22.26	47.46	21.10	47.77	21.67	45.90	20.82	49.10	22.27	46.76	21.21
8	53.92	24.46	52.04	23.44	52.42	23.78	50.44	22.88	53.81	24.41	50.87	23.07
9	59.23	26.87	57.07	25.91	57.45	26.06	55.29	25.08	59.46	26.97	56.44	25.60
10	65.30	29.62	62.35	28.29	62.43	28.32	60.60	27.49	65.35	29.64	62.45	28.33
11	70.18	31.84	68.84	31.23	68.34	31.00	66.47	30.15	70.92	32.17	68.84	31.22
12	76.92	34.89	78.31	35.53	73.73	33.51	74.21	33.66	76.08	34.51	77.82	35.30
13	84.84	38.49	88.65	40.21	80.71	36.61	84.85	38.49	84.89	38.50	87.96	39.90
14	94.91	42.95	98.43	44.65	89.15	40.44	93.23	42.29	95.76	43.43	97.64	44.29
15	107.10	48.59	106.08	48.12	101.90	46.22	102.93	46.69	109.05	49.46	105.87	48.02
16	121.01	54.90	112.03	50.81	113.76	51.60	110.78	50.25	122.06	55.36	110.58	50.16
17	127.49	57.84	115.53	52.41	122.73	55.67	115.98	52.61	130.35	59.12	113.32	51.40
18	132.55	60.13	115.16	52.24	137.76	62.49	112.48	51.02

¹ 6th Ann. Rep. State Board of Health, Wisconsin, 1881, p. 28.

² Jahrb. f. Kinderheilk., 1901, LIII, 53.

³ Loc. cit., 306.

lb. (3674) or 8.15 per cent. for boys and 7.5 lb. (3402) or 6.76 per cent. for girls.

The influence of any illness upon the increase of weight is very decided, especially in infancy. Growth ceases, and weight may even be rapidly lost, depending upon the nature of the disease. There appears to be a positive influence of *season* upon the growth of children. The investigations of Malling-Hansen¹ upon children of from 9 to 17 years showed the most rapid increase occurring from August to December. Schmidt-Monard² confirms this for even younger children from the age of 2½ years and even in the 2d year, and Bleyer³ found it true even of the first year of life. Indeed the influence of the season may extend so far that, as claimed by Stepanoff,⁴ children of the school age who were born in summer are larger than those born in winter.

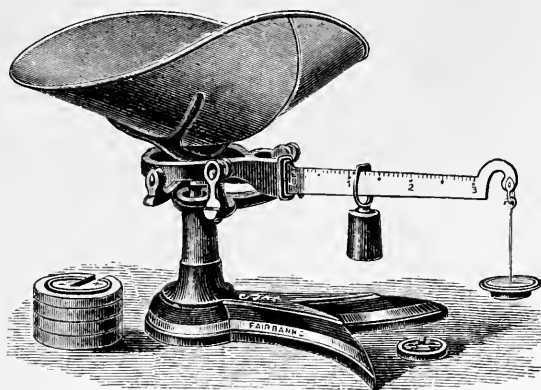


FIG. 4.—BALANCE SCALES FOR WEIGHING INFANTS.

The importance of weighing children regularly during the first 2 years of life, and especially during the 1st year, cannot be over-estimated, since cessation of gain is often the first sign of illness, or of a lack of sufficient nourishment. The child should be weighed at least weekly, divested of clothing or dressed and the weight of the clothes subsequently deducted. Scales should be used which indicate ounces. Standing spring scales with a scoop or basket attached are very convenient, but some form of balance scale is more accurate (Fig. 4). For recording the results a *weight chart* is a great convenience. The illustration (Fig. 5) is a reduced reproduction of one which I have employed for a number of years.⁵ The curve already plotted upon it represents the normal average gain in weight for a healthy breast-fed infant. To economize space the portion for the 2d year is narrowed one-half, which necessarily distorts the line. A very convenient form for recording daily weighings is that employed at The Children's Hospital, Philadelphia, of which a reduced reproduction is shown (Fig. 6).

Length.—Estimations upon growth in length in the first year have not been nearly so numerous as those upon weight, and the results of

¹ *Perioden in Gewicht, etc.*, Copenhagen, 1886. Ref. Vierordt's *Daten u. Tabellen*, 1906, 25.

² *Loc. cit.*

³ *Arch. of Pediat.*, 1917, XXXIV, 366.

⁴ *Thèse de Lausanne*. Ref. *Monatsschr. f. Kinderheilk.*, 1903, II, 242.

⁵ W. B. Saunders Co. For sale singly by E. Pennoek, 3609 Woodland Ave., Phila.

different observations show decided variations, since accurate determination requires much care. The average length at birth may be assumed as 49.5 to 50 cm. (19.5 to 20 inches), with males about 0.5 to

INFANTS' WEIGHT CHART.

Drawn by J. P. CROZER-GRIFFITH, M. D.,
Clinical Professor of Diseases of Children at
the University of Pennsylvania.

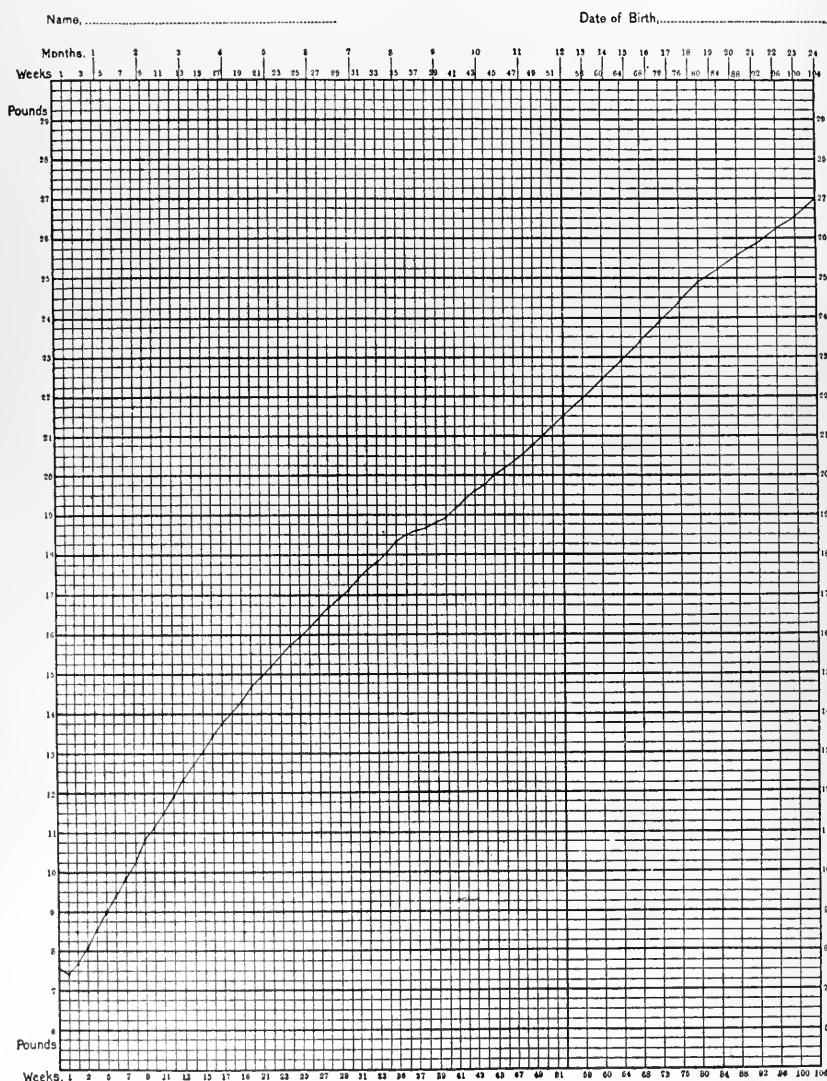


FIG. 5.—AUTHOR'S WEIGHT CHART FOR THE FIRST 2 YEARS OF LIFE.

Reduced size. (*New York Med. Journ.*, 1899, March 4.)

1 cm. (0.2 to 0.4 inch) longer than females. Gain in length goes on rapidly but with decreasing speed. Thus in the first 3 months, according to Camerer,¹ the gain is 9 cm. (3.5 inches), in the next 3 months 8 cm.

¹ *Jahrb. f. Kinderh.*, 1901, LIII, 425.

(3.2 inches), and in the third and fourth 3-month periods 3 to 5 cm. (1.2 to 2 inches), making a total gain of 24 cm. (9.4 inches) for the year. These figures are somewhat in excess of certain other statistics. Heubner¹

INFANT'S WEIGHT CHART

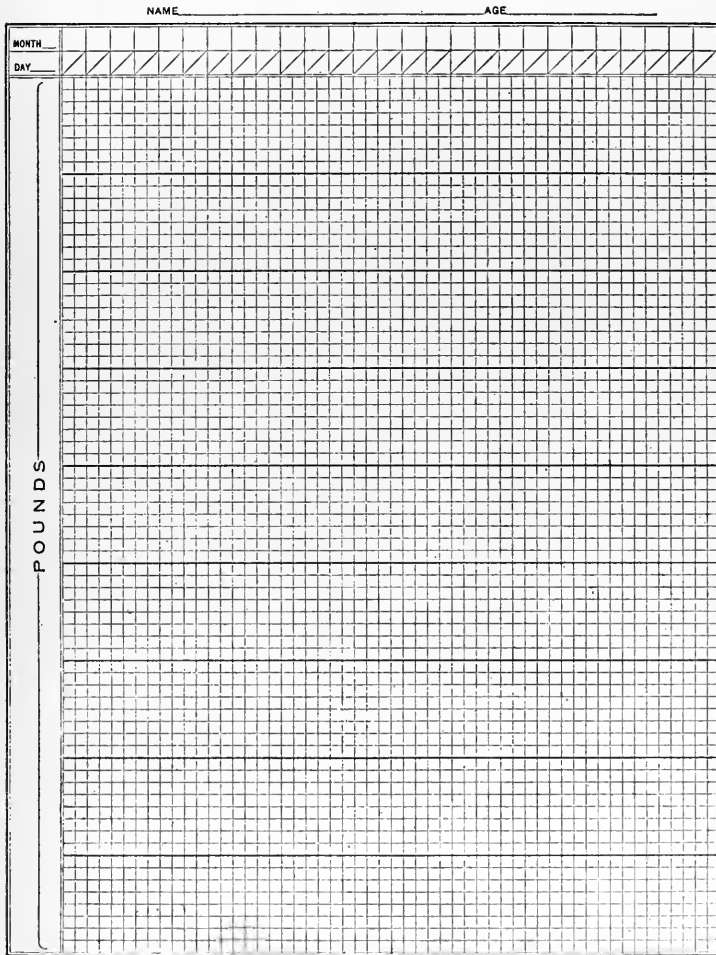


FIG. 6.—DAILY WEIGHT CHART.

To be used for daily weighings at any period of infancy. As employed in the Children's Hospital and in the Children's Medical Ward of the University Hospital, Philadelphia. Reduced size.

gives the following table for average figures taken from different investigators:

As the table shows, the length of male children continues slightly in excess of that of females, the differences at the end of the year equalling about 1 cm. (0.4 inch).

¹ Lehrb. d. Kinderh., 1911, I, S.

TABLE 9.—GAIN IN LENGTH IN BOYS AND GIRLS
(Heubner)

	Boys	Girls
Birth.....	51.0 cm. (20.1 in.)	49.0 cm. (19.3 in.)
1 month.....	52.5 cm. (20.7 in.)	51.3 cm. (20.2 in.)
2 months.....	55.3 cm. (21.8 in.)	54.8 cm. (21.6 in.)
3 months.....	57.8 cm. (22.7 in.)	56.7 cm. (22.3 in.)
4 months.....	60.1 cm. (23.7 in.)	58.7 cm. (23.1 in.)
5 months.....	61.3 cm. (24.1 in.)	60.2 cm. (23.7 in.)
6 months.....	62.6 cm. (24.6 in.)	61.5 cm. (24.2 in.)
7 months.....	64.6 cm. (25.4 in.)	63.2 cm. (24.9 in.)
8 months.....	65.6 cm. (25.8 in.)	64.3 cm. (25.3 in.)
9 months.....	67.8 cm. (26.7 in.)	65.4 cm. (25.8 in.)
10 months.....	67.0 cm. (26.4 in.)	67.2 cm. (26.5 in.)
11 months.....	69.0 cm. (27.2 in.)	68.1 cm. (26.8 in.)
12 months.....	70.3 cm. (27.7 in.)	69.2 cm. (27.2 in.)

During the periods covered by the ages from 1 to 5 years the gain in the 2d year of life is about 10 cm. (3.9 inches); in the 3d, 9 cm. (3.5 inches) and in the 4th and 5th years 7 cm. (2.8 inches). The following figures show this in tabular form, assuming the length at 1 year to be 28 inches (71 cm.):

TABLE 10.—GAIN IN LENGTH IN THE FIRST FIVE YEARS

1 year.....	28.0 in. (71 cm.)
2 years.....	32.0 in. (81 cm.)
3 years.....	35.5 in. (90 cm.)
4 years.....	38.25 in. (97 cm.)
5 years.....	41.0 in. (104 cm.)

The studies of Freeman¹ upon 278 well-cared-for children in private practice showed a length for the first 5 years decidedly above these figures. This depends doubtless on the patients being of a better social class.

During this period the difference in length between the sexes continues, but does not increase.

From the age of 5 years onward the combined rate of increase in the two sexes is about 2 inches (5 cm.) a year until the age of 11 years, after which girls slightly exceed boys in height until the age of 15 years, when boys again take the lead. Increase in length is very slight in girls after they reach 16 years. The length at birth is doubled between the ages of 4 and 5 years and trebled at about 13 or 14 years.

The following table shows the gain in height in boys and girls respectively, according to the extensive observations of Bowditch,² Porter,³ Peckham⁴ and Variot and Chaumet.⁵ Very similar statistics in both height and weight are given by Tuxford and Glegg.⁶

Growth in length exhibits certain definite seasonal relationships, analogous to, though differing from, those affecting that in weight. The greatest increase in length, according to the studies of Schmidt-Monard⁷ in children from 2 to 13 years, takes place in July and August,

¹ Amer. Jour. Dis. Child., 1914, VIII, 321.

² Loc. cit.

³ Loc. cit.

⁴ Loc. cit.

⁵ Bul. soc. de p  d. de Paris, 1906, Feb., 53.

⁶ Brit. Med. Jour., 1911, I, 1423.

⁷ Jahrb. f. Kinderheilk., 1895, XI, 84.

On the other hand while the legs of the girl at 9 years are longer than those of the boy, the boy much surpasses her later in length of leg. In fact, girls increase very little in length of leg after the age of 14 or 15 years, while boys grow both in legs and trunk.

General Surface.—By the age of 2 weeks the dark-red color of the skin of the body has changed to the rose-pink characteristic of infancy. Generally by the end of the first week or earlier any lanugo remaining begins to fall, and this is accompanied by a more or less extensive shedding of the epidermis, sometimes in small scales, sometimes in large flakes. This shedding lasts from 2 to 3 weeks. The sudoriparous glands are not very active in the first 4 weeks of life in healthy children. Very soon, however, they secrete well and the insensible and even sensible perspiration is considerable, although free perspiration normally does not occur for several months.

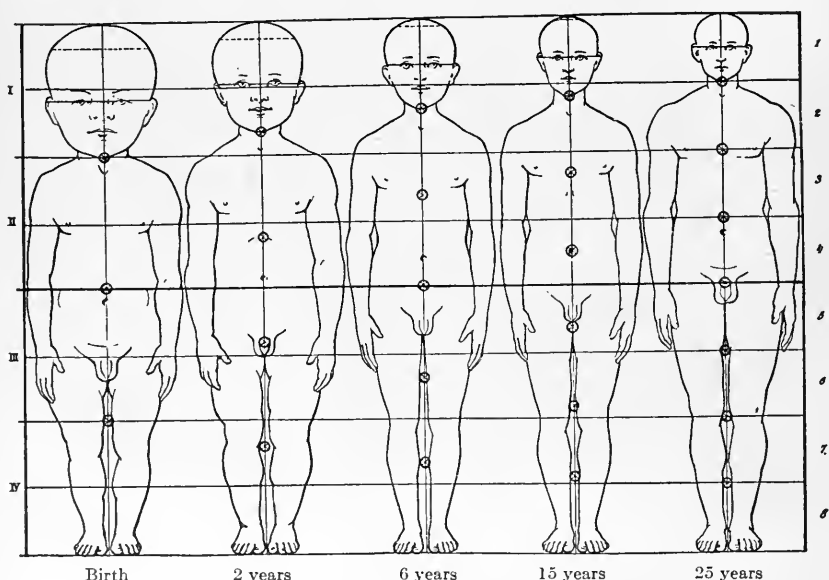


FIG. 7.—GROWTH-PROPORTIONS AT DIFFERENT LIFE-PERIODS.

Showing the length of different portions of the body as compared with the head at different periods of life. At birth the body is 4 heads high, while in the adult it is 8 heads high. (After Stratz, *Der Körper des Kindes*, 1904, 64, Fig. 42.)

Head. Size and Shape.—The *circumference* of the head at birth averages about 13 to 13 $\frac{3}{4}$ inches (33 to 35 cm.). The measurement in new-born girls is from 0.2 to 0.4 inch (0.5 to 1 cm.) less than in boys. Growth is at first rapid, but is very slow after the age of 5 years. Published statistics vary considerably, but a comparison of a number of investigations gives the following average figures of the circumference at birth and of the increase:

TABLE 12.—CIRCUMFERENCE OF THE HEAD

Birth.....	33 to 35 cm. (13.0 to 13.8 in.)
6 months.....	42 to 45 cm. (16.5 to 17.7 in.)
1 year.....	45 to 46 cm. (17.7 to 18.1 in.)
2 years.....	47 to 48 cm. (18.5 to 18.9 in.)
3 years.....	48.5 to 50 cm. (19.1 to 19.7 in.)
4 years.....	50 to 52 cm. (19.7 to 20.5 in.)
5 years.....	52 to 53 cm. (20.5 to 20.9 in.)
Adult.....	53 to 55 cm. (20.9 to 21.7 in.)

It will be noticed that the growth in circumference is about 10 cm. (3.9 inches) during the 1st year, which is approximately one-half of the increase of the child in length. In the 2d year the increase is about 2 cm. (0.8 inch). Girls gradually fall behind to the amount of $1\frac{1}{2}$ to 3 cm. (0.6 to 1.2 inches) in the circumference of the head.

The *shape* of the head differs decidedly in infants from that in adult life (Fig. 8). The facial portion is small as compared with the cranial; the ratio of the respective dimensions in the new born being 1 : 8 and in the adult 1 : 2 or $2\frac{1}{2}$ (Froriep).¹ Its rate of growth is, however, more rapid (Weissenberg).² The caput succedaneum, *i.e.*, the swelling of the scalp

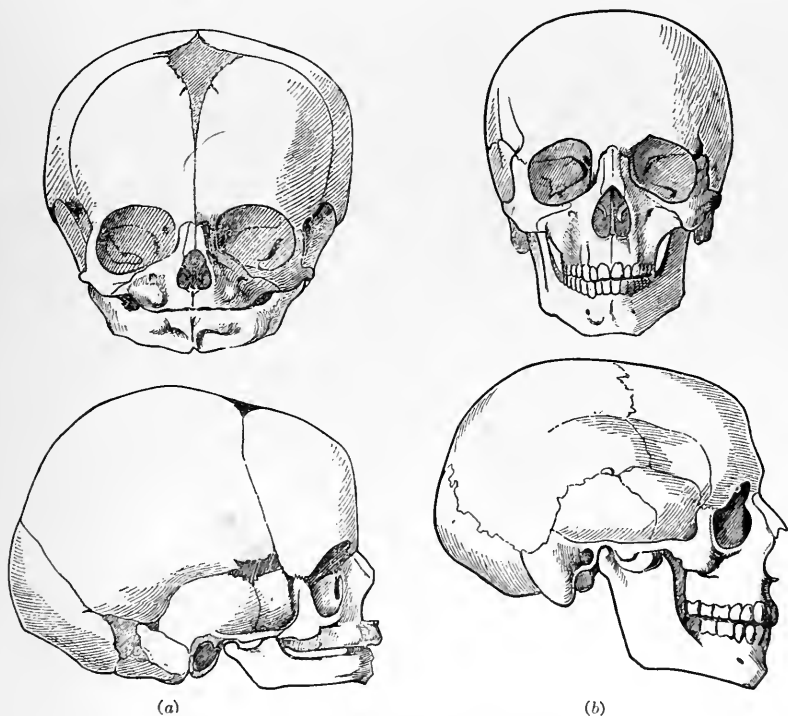


FIG. 8.—COMPARISON OF SKULLS.

Of the infant (a) and of the adult (b). (After Henke, Gerhardt's *Handb. d. Kinderkr.*, I, 248; 249.)

resulting from pressure during labor, generally disappears by the 10th day or earlier (see p. 271.)

Owing to the yielding nature of the bones of the skull and the absence of union of the sutures the shape may be altered by long-continued pressure as by that during labor, or by the child lying too much upon the back or side, thus flattening the occiput or one of the parietal regions. This distortion is usually only of temporary duration.

Fontanelles.—The posterior fontanelle can be felt open until the age of 6 or 8 weeks. The anterior, of rhomboidal shape, increases in size according to the views of most observers until the 9th month, owing to

¹ Die Charakteristik des Kopfes nach dem Entwicklungsgesetze desselben. Berl., 1845. Ref. Henke, Gerhardt's *Handb. d. Kinderkr.*, 1877, I, 250.

² *Jahrb. f. Kinderh.*, 1908, LXVIII, 316.

the fact that the brain grows faster than the bones covering it. By the 12th month it should be decidedly smaller. According to Kassowitz,¹ however, it should grow steadily smaller from the time of birth, and any increase in its size must be attributed to rickets. The fontanelle should be closed by the age of 17 or 18 months. Even in healthy children it sometimes closes earlier or remains open longer than the average time allows, but closure should be accomplished by 2 years at the latest. It is slightly larger in boys, and in the children of primiparæ (Fehling).² There are great variations in the size of the anterior fontanelle at birth, and in the statements made regarding this. A diameter ranging from 2 to 2½ cm. (0.8 to 1 inch) between the parallel sides perhaps expresses the average. Elsässer³ gives the following figures for the changes which take place with time:

TABLE 13.—SIZE OF ANTERIOR FONTANELLE
(Elsässer)⁴

1 to 3 months.....	2.51 cm. (1.0 in.)
4 to 6 months.....	3.12 cm. (1.2 in.)
7 to 9 months.....	3.63 cm. (1.4 in.)
10 to 12 months.....	3.11 cm. (1.2 in.)
13 to 15 months.....	2.03 cm. (0.8 in.)

Scalp.—Although the scalp at birth frequently shows only short sparse hair it is often covered with a thick, rather dark, comparatively long growth, measuring from 1½ to 2 inches (3.8 to 5.1 cm.). This begins to fall at the end of the first week and generally leaves the head almost bald, although some stays on much longer than this, and many infants retain a heavy growth of hair for months. The new hair comes in only slowly, and is of firmer texture and generally lighter color. Unless precautions are taken a collection of oily scales very rapidly accumulates on the scalp.

Eyes.—The eyes at birth and in the first weeks of life are kept shut or half open and are largely devoid of expression. Their color is a somewhat indefinite blue-gray, which only later changes into the permanent hue of the iris, varying in different subjects. The movements of the eyes are largely incoördinated. Tears are usually not shed until the age of 3 or 4 months.

Ears.—The meatus is not fully developed at birth, the portion finally osseous not becoming so until the 4th year. The direction of the meatus is inward and downward and the tympanic membrane is horizontal or inclined slightly upward. The tympanic cavity is devoid of air at birth and contains only swollen mucous membrane and mucus. The **nose** of the new born is relatively small and, as a whole, situated higher in the face than in adult life. The maxillary antrum and ethmoidal cells are present at birth; the other sinuses develop later (Coffin).⁵ The fat in the **cheeks** is comparatively well developed, and forms in each what is called the "sucking-cushion." The **jaws** are small and in a rudimentary condition at birth. The angle of the ramus and body of the lower jaw is much more obtuse than later in life. During infancy the jaws

¹ Verhandl. d. deutsch. Gesellsch. f. Kinderheilk., Strassburg, 1885.

² Arch. f. Gynäk., 1875, VII, 575.

³ Die weiche Kinderkopf, 1843, 10.

⁴ I have used the metric equivalents for Elsässer's statistics as given by Vierordt (Daten und Tabellen, 1906, 104). The original is in "the old Parisian lines" (p. 9).

⁵ Amer. Jour. Med. Sci., 1905, CXXIX, 302.

grow considerably, but especially in later childhood they enlarge in order to admit the permanent molar teeth.

Spine.—The spinal column of the new born is of such flexibility that the existence of any of the natural curves of later life is doubtful. The sacrococcygeal curve is present, it is true, but the remainder of the spine either forms one long continuation of this or is straight. Symington¹ has pointed out that the *neck*, as compared with the rest of the spinal column, is in reality relatively longer than in adult life, about equalling the lumbar portion. Its apparent shortness is due to the large amount of fat covering it, and to the high position of the sternum. The curve in the neck, with the convexity forward, does not appear until the child begins to hold its head erect, and never becomes fixed. Still later, when the child learns to stand and walk, a similar curve forms in the lumbar region and one with the concavity forward in the dorsal region. The lumbar spine grows faster than the other portions until a little after puberty.

Thorax.—The chest in infancy is small as compared with the abdomen and with the thoracic development of later life. The combination of small chest and large abdomen, together with the high position of the narrow and insignificant shoulder-girdle, gives the trunk a peculiar barrel-shaped appearance. The *nipples* are small and are situated in the 4th interspace or over the 4th rib as in adult life.

The *ribs* are more horizontally placed than in adult life and the false ribs particularly project upward to a greater extent. The *diaphragm* extends somewhat higher, and the *sternum* is relatively smaller than in adult life. The upper border of the manubrium of the sternum stands higher than later while the lower projects more sharply forward.

Whereas in adults the transverse diameter of the thorax is to the anteroposterior as 2:1 (Fetzer),² or 3:1 (Symington)³ in the new born the diameters according to Eckerlein⁴ are nearly equal, the transverse being to the antero-posterior as 3:2. As a result the horizontal section of the thorax in early infancy appears nearly circular, while that of the adult is elliptical. The transverse diameter grows more rapidly than the other, and the adult shape of the chest is present to a large degree by the beginning of childhood.

There is considerable diversity in the estimates of different investigators regarding the circumference of the chest at birth taken at the height of the nipples, and the rate of growth in the first 5 years of life. This depends upon the great variation to which this growth is susceptible, rendering any statistics only average ones. Approximate figures, the average obtained from the statistics of a number of authors, read as follows:

TABLE 14.—GROWTH OF CHEST IN THE FIRST FIVE YEARS

Birth.....	32 to 33 cm. (12.6 to 13.0 in.)
6 months.....	41 to 42 cm. (16.1 to 16.5 in.)
1 year.....	44 to 46 cm. (17.3 to 18.1 in.)
2 years.....	45 to 48 cm. (17.7 to 18.9 in.)
3 years.....	50 to 51 cm. (19.7 to 20.1 in.)
4 years.....	52 to 53 cm. (20.5 to 20.9 in.)
5 years.....	54 to 56 cm. (21.3 to 22.0 in.)

¹ Anatomy of the Child, 1887.

² Ueber die Einfluss d. Militärdienstes auf d. Körperentwicklung, 1879, 198; Ref. Vierordt's Daten u. Tabellen, 1906, 98.

³ Anat. of the Child, 1887.

⁴ Zeitsch. f. Geburtsh. u. Gyn., 1890, XIX, 120.

In female children the circumference is from 0.5 to 1.5 cm. (0.2 to 0.6 inch) less than in male.

The difference in the rate of growth of the thorax in each sex after the age of 5 years up to that of 18 years is illustrated by the observations of Porter¹ in over 34,000 measurements of school children in St. Louis, Mo., and given in the following table:

TABLE 15.—GROWTH OF CHEST IN BOYS AND GIRLS

Years	Chest	
	Boys	Girls
6	59.05 cm. (23.2 in.)	58.34 cm. (23.0 in.)
7	60.62 cm. (23.9 in.)	59.47 cm. (23.4 in.)
8	62.18 cm. (24.5 in.)	60.81 cm. (23.9 in.)
9	63.90 cm. (25.2 in.)	62.51 cm. (24.5 in.)
10	65.59 cm. (25.8 in.)	63.02 cm. (24.8 in.)
11	67.24 cm. (26.5 in.)	65.85 cm. (25.9 in.)
12	68.76 cm. (27.1 in.)	68.34 cm. (26.9 in.)
13	70.61 cm. (27.8 in.)	71.29 cm. (28.1 in.)
14	73.27 cm. (28.9 in.)	74.13 cm. (29.2 in.)
15	76.56 cm. (30.1 in.)	76.78 cm. (30.6 in.)
16	79.22 cm. (31.2 in.)	78.85 cm. (31.0 in.)
17	81.39 cm. (32.0 in.)	80.39 cm. (31.7 in.)
18	84.52 cm. (33.3 in.)	80.45 cm. (31.7 in.)

According to the observations of Monti² the average increase in circumference in the 1st year is 12 cm. (4.7 inches) in the 2d year 3 cm. (1.2 inches) and in the remaining years up to 12, 1.25 cm. (0.5 inch). There then occurs a sudden increase in the rate of growth, averaging 4 cm. (1.57 inches) a year.

The comparison of the rate of growth in the circumference of the chest and of the head respectively is of great practical utility. The combination of the two tables already presented (pp. 32 and 35) gives the following results:

TABLE 16.—COMPARISON OF CIRCUMFERENCES OF THE HEAD AND CHEST

	Head	Chest
Birth.....	33-35 cm. (13.0 to 13.8 in.)	32-33 cm. (12.6 to 13.0 in.)
6 months.....	42-45 cm. (16.5 to 17.7 in.)	41-42 cm. (16.1 to 16.5 in.)
1 year.....	45-46 cm. (17.7 to 18.1 in.)	44-46 cm. (17.3 to 18.1 in.)
2 years.....	47-48 cm. (18.5 to 18.9 in.)	45-48 cm. (17.7 to 18.9 in.)
3 years.....	48.5-50 cm. (19.1 to 19.7 in.)	50-51 cm. (19.7 to 20.1 in.)
4 years.....	50-52 cm. (19.7 to 20.5 in.)	52-53 cm. (20.5 to 20.9 in.)
5 years.....	52-53 cm. (20.5 to 20.8 in.)	54-56 cm. (21.3 to 22.0 in.)

This relationship is, however, open to considerable variation. The chest often grows quite rapidly, its circumference equalling that of the head by the age of 1 year. Still Frobelius³ has found from a large number of measurements, that the circumference of the chest at birth should never be over 2 to 2.5 cm. (0.8 to 1 inch) less than that of the head, and that

¹ Transac. Acad. Science St. Louis, VI, No. 12, 354.

² Kinderheilk. in Einzeldarst., 1899, I, 565.

³ St. Petersburg. med. Zeitsch., 1873, IV, 363.

in proportion as these figures are exceeded the mortality is much increased. It is certainly a sign of feeble development when the circumference of the chest has not exceeded that of the head by the age of 3 years.

Abdomen.—The abdomen appears relatively large and prominent in infancy, depending in part upon the large size of the liver, in part on the great amount of subcutaneous fat present, and in part on the small size of the pelvis and of the chest. Its circumference measures about the same as that of the chest up to 2 years after which period it is decidedly less. The stump of the *umbilical cord* remaining after birth gradually shrivels, exhibiting a small red area around it at its junction with the body. About the 4th to the 6th day the stump falls, leaving an ulcer which heals rapidly, closing after 5 to 6 days. The umbilicus occupies nearly the central part of the body during the first 2 years. In adult life its distance above the soles is $\frac{3}{5}$ of the total length of the body.

Pelvis.—The pelvis in the infant is very small and more obliquely situated than later. According to Liharzik,¹ the width of the body at the hips at all ages equals that of the shoulders in males, but the hips are always wider than the shoulders in females.

Limbs.—The bowing of the short *legs* present at birth, which depends upon an actual curve in the bone, persists until during the 2d year. The tendency to hold the arms and legs in the fetal position (p. 19), with the feet dorsally flexed, is exhibited more or less during the early months. Although infants are seemingly flat-footed, in reality the feet are shaped much as in adult life, the shortness and thickness seen especially in later infancy depending in reality upon the large amount of fat in the subcutaneous tissue.

DIGESTIVE APPARATUS

Mouth.—The mouth in early infancy is comparatively dry with the tongue also drier than later and generally with a whitish coating. The jaws at birth will not meet. They are covered with gums of a pale-red color, and exhibit rather hard narrow opposing ridges. No signs of the presence of teeth are visible.

Dentition.—The 20 *temporary teeth*, also called *deciduous*, *first*, or *milk teeth*, that appear later are at birth enclosed in dental follicles, and in alveoli in the jaw which are already osseous in nature. The crowns of the incisor teeth are even then entirely calcified, as are those of the other teeth to a considerable extent. The teeth and tooth-sacs are covered only by mucous membrane and sub-mucous connective tissue, and the alveoli are broad and allow of free growth. As calcification and elongation of the roots take place, the crowns are gradually forced onward in the direction of least resistance. Under this constant pressure outward the gums covering them atrophy, flatten and grow paler, and the teeth finally push through. Probably the only resistance offered has come from the sub-mucous tissue. Complete calcification of the fangs is not present when the teeth appear, and does not occur until the child is several years of age.

The germs of the permanent teeth, or second teeth, except the second and third molars, are already present in the gums at birth, resting against

¹Das Gesetz des Wachstums, etc., 1862; Ref. Vierordt's Daten u. Tabellen, 1906, 30.

the posterior walls of the dental sacs of the temporary teeth. The crowns of the anterior molars are calcified. In the other teeth of this set the calcification begins at different periods between the first and the eighth year. Like the first set they increase in length, their eruption depending principally upon the calcification of the roots. Probably from insufficient supply of blood, occasioned by pressure of the advancing permanent teeth, the roots of the temporary set finally undergo absorption and the teeth drop out in much the same order as they came in.

Eruption of the Temporary Teeth (Fig. 9).—Many different statements have been made by authors regarding the time of the eruption of the 20 temporary teeth. The following table expresses a very generally accepted view. It will be noticed that the teeth erupt in distinct groups, with a period between each group.

TABLE 17.—ERUPTION OF THE TEMPORARY TEETH

First Group.....	2 lower central incisors.....	7 months
	Pause.....	3 to 8 weeks. Total 2.
Second Group.....	4 upper incisors.....	8 to 10 months
	Pause.....	1 to 3 months. Total 6.
Third Group.....	4 anterior molars and 2 lower lateral incisors...	12 to 15 months
	Pause.....	2 to 3 months. Total 12.
Fourth Group.....	4 canines.....	18 to 24 months
	Pause.....	2 to 4 months. Total 16.
Fifth Group.....	4 posterior molars.....	20 to 30 months
		Total 20.

The upper and lower canines are popularly called the “eye-teeth” and the “stomach teeth” respectively. In the third group the lower lateral

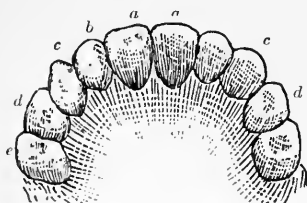


FIG. 9.—DIAGRAM SHOWING THE TEMPORARY TEETH.

a, central incisors; b, lateral incisors; c, canines; d, anterior molars; e, posterior molars.

incisors appear, according to many observers, before the molars, but there is no absolute rule for this. In fact the whole table represents only the order of eruption very frequently seen, and it is certain that there may be an extremely wide variation within physiological limits. The age of 7 months may be considered the average time of appearance of the first tooth, yet any time not exceeding the 1st year may be called normal. From 6 to 8 months is a very common range, and eruption at 3 or 4 months is not at all uncommon.

In perfectly normal states the eruption of the teeth is unattended by any symptoms whatever. The pathological conditions which are believed to develop will be considered in the section upon Disorders of Dentition (p. 651).

Eruption of the Permanent Teeth (Fig. 10).—The *permanent teeth*, or *teeth of the second dentition*, are 32 in number. The earliest to be cut are the first molars, which come in just posterior to the temporary second molars. They appear at about the age of 6 years and are consequently often called the “6-year-old molars.” The other teeth erupt in much the same order as those of the temporary set. Although the order and dates of appearing are subject to considerable variation, the following table is a fair expression of them:

TABLE 18.—ERUPTION OF THE PERMANENT TEETH

First molars.....	6 years
Central incisors.....	7 years
Lateral incisors.....	8 years
First bicuspid.....	9 years
Second bicuspid.....	10 years
Canines.....	11 to 13 years
Second molars.....	12 to 15 years
Third molars (Wisdom teeth).....	17 to 25 years

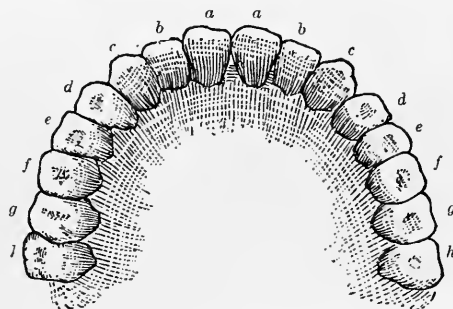


FIG. 10.—DIAGRAM SHOWING THE PERMANENT TEETH.

a, central incisors; b, lateral incisors; c, canines; d, first bicuspid; e, second bicuspid; f, first molars; g, second molars; h, third molars.

The teeth of the lower jaw usually erupt somewhat before the corresponding ones of the upper jaw, the interval being often as much as several months. It will be observed that the permanent molars do not replace any of the temporary teeth, but constitute 12 new teeth additional to the original 20. The bicuspid replace the temporary molars



FIG. 11.—JAWS OF CHILD ABOUT 7 YEARS OLD, SHOWING TEMPORARY AND PERMANENT SETS OF TEETH, EXCEPT THE WISDOM TEETH.

(McClellan, in Keating's *Cyclopedia of the Diseases of Children*, 1889, I, 18, Fig. 12).

and the canines and incisors the corresponding teeth of the first set. The bicuspid are smaller than the temporary molars, and the permanent incisors larger than those which preceded them. At about the age of 6 years the jaws contain all the teeth of both sets, visible or concealed, except the third permanent molars (Fig. 11).

Salivary Glands.—The comparatively small amount of saliva secreted by the new born accounts for the dryness of the mouth and the coating of the tongue referred to. By the 2d month increase in the secretion begins and by the age of 3 or 4 months this is still further augmented and the child begins to "dribble." This increase takes place, as a rule, before the eruption of teeth, and appears to be in no way connected with it. Although according to the investigations of Korowin¹ and of Zweifel² ptyalin is present in the salivary glands even at birth, any diastasic activity is inconsiderable until the increased secretion of saliva begins. Even then, since much of the fluid runs out of the mouth, its influence upon digestion is probably not great. As the first year advances the amount of saliva grows greater and by about the age of 1 year its diastasic power is about as great as in adult life (Finizio).³

Esophagus.—The total length of the esophagus in the new born, according to the statistics collected by Vierordt,⁴ is about 10 cm. (3.9 inches) or, including the total distance from the teeth to the cardiac orifice, 17 cm. (6.7 inches). This latter distance at the age of 3 years is 30 cm. (11.8 inches) and in adult life 40 cm. (15.8 inches). The pseudo-valvular opening at the gastric end of the esophagus is stated by Gubiaroff⁵ to be imperfectly developed in infancy. This accounts in part for the greater ease with which vomiting occurs at this time of life.

Stomach.—The capacity of the stomach varies greatly in different infants within normal limits, as is shown by the results obtained by different observers. This is illustrated in the following table from which, however, an idea of the approximate size may be obtained:

TABLE 19.—CAPACITY OF THE STOMACH AT DIFFERENT PERIODS*

	Fleischmann ⁶		Holt ⁷		Frolowsky ⁸		Pflaunder ⁹		Beneke ¹⁰	
	C.c.	Fl.oz.	C.c.	Fl.oz.	C.c.	Fl.oz.	C.c.	Fl.oz.	C.c.	Fl.oz.
Birth.....	36	1.2	35-43	1.2-1.5
1 week.....	46	1.6	50	1.7	153-160	5.2-5.4
2 weeks.....	72	2.4	44	1.5	70	2.4		
1 month.....	80	2.7	59	2.0	112	3.8	90	3.0		
2 months.....	140	4.7	100	3.37	158	5.3	100	3.4		
3 months.....	133	4.50	167	5.6	110	3.7	740	25
4 months.....	148	5.00	178	6.4	125	4.2		
6 months.....	176	5.75	160	5.4		
9 months.....	253	8.6	225	7.6		
1 year.....	263	8.9	290	9.8		
2 years.....		

* The original tables vary slightly from this, the data being for the number of weeks rather than of months. As the differences, however, are inconsiderable, I have purposely distorted the figures slightly to apply to the age in months.

¹ Centralb. f. d. med. Wissensch., 1873, XI, 261.

² Untersuch. ü. d. Verdauungsapparat der. Neugeb., 1874.

³ Rev. d'hyg. et de méd. inf., 1909, VIII, 224.

⁴ Daten u. Tabellen, 1906, 112.

⁵ Arch. f. Anat. u. Entwicklungsgeschichte, 1886, 395. Ref. Jacobi, Keating's Cyclop. of the Dis. of Child., 1889, I, 35.

⁶ Klinik d. Pädiatrik, 1875, I.

⁷ Arch. of Ped., 1890, 963.

⁸ St. Petersb. Dissertation, 1876. Ref. Vierordt, Daten u. Tabellen, 1906, 115.

⁹ Wien. klin. Wochenschr., 1897, No. 44.

¹⁰ Deut. med. Wochenschr., 1880, VI, 448.

Roughly we may estimate the capacity of the stomach at birth at $1\frac{1}{4}$ fl.oz. (37), at 1 month as $2\frac{1}{2}$ fl.oz. (74), at 6 months as 5 fl. oz. (148), and at 1 year as $9\frac{3}{4}$ fl.oz. (288). It depends, however, on the size of the child as much as or more than on the age alone. This must always be taken into consideration in determining the amount of food required by an infant of a certain age. On the other hand, it is also true that an infant not infrequently can digest, and requires, a larger amount of food than its age would indicate. This may well be because some of the liquid taken passes into the intestine before the meal is completed. Basing the dietetic requirements upon the estimated size of the stomach is consequently a procedure to be depended upon only with limitations. The capacity in artificially fed infants is somewhat greater than in those fed at the breast. The orifice of the pylorus measures 2 cm. (0.78 inch) in circumference in the new born, according to Pflaunders.¹ The position of the stomach early in infancy had been generally considered to be nearly vertical or slightly oblique, and the form cylindrical; but the more recent investigations are not entirely in accord with this. Fleisch and Péteri² found it nearly horizontal in the 1st year of life, but becoming more vertical after this period. Smith³ believes it to be not vertically situated, but rather obliquely, and Pisek and LeWald⁴ showed that the organ changes its size and shape from time to time, depending upon the amount of food contained in it. It is oftener horizontally situated rather than vertically, and the pylorus generally occupies a comparatively high and anterior position. The amount of gastric secretion is large in infancy. Pepsin, rennin and hydrochloric acid are present at all periods, although the amount of rennin and of acid occurring in the new born is proportionately much less than in adult life (Leo).⁵ The amount, however, rapidly increases as the infant grows older. According to Langendorf⁶ pepsin is found in the fetus even as early as the 4th month of intra-uterine life. It is possible that rennin is not a substance distinct from pepsin. A fat-splitting ferment is also present, as demonstrated by Sedgwick⁷ and others; and Ibrahim and Kopec⁸ found it in the 6-months' fetus.

Liver.—The liver at birth is hyperemic and large and its edge can be distinctly felt below the costal margin. To a great extent it covers the anterior and outer surface of the stomach. It continues to extend below the costal margin until about the age of 5 years. The area of hepatic dullness is also greater in early life, the upper margin of this reaching the 4th rib. According to the statistics collected by Vierordt⁹ its absolute weight averages 141.7 grams (5 oz.) in the male and 164 grams (5.8 oz.) in the female; *i.e.*, 4.5 per cent. and 5.5 per cent. of the body-weight, while in the adult it is but from 2.8 per cent. to 3 per cent. The weight increases little or none during the first 6 months, and, in fact, diminishes slightly in the first weeks. Kowalski's¹⁰ figures, however,

¹ Ueber Magencapazität, etc., Bibliotheca medica, 1898, 35.

² Zeit. f. Kinderh., Orig., 1911, II, 263.

³ Arch. of Ped., 1914, XXXI, 784.

⁴ Amer. Jour. Dis. Child., 1913, VI, 232.

⁵ Berl. klin. Wochenschr., 1888, 931.

⁶ Arch. f. Physiolog., 1879, 95.

⁷ Arch. of Ped., 1906, XXIII, 414.

⁸ Zeitsch. f. Biol., 1910, LIII, 201.

⁹ Arch. f. Anat. u. Physiol., Suppl. Band, 1890, 62.

¹⁰ Dissert. St. Petersburg, 1908, 21. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1915, 15.

do not show this failure to increase in weight in the early months. Bile is secreted even before birth. The investigations of Jacobowitsch¹ indicate that the bile of the infant differs from that of the adult especially in the larger proportion of water and the smaller amount of biliary acids and of bile-salts present, and the consequent lesser power of digesting the fats.

Pancreas.—The pancreas at birth weighs 3.5 grams (0.12 oz.) (Vierordt);² bearing about the same relation to the body-weight as in adult life. Its secretion has the power of digesting fat in early infancy, but not to the degree possessed later. The diastatic power (amyllopsin) was claimed by Korowin³ to be absent during the first 3 weeks, feeble after this, and not to attain its full power until about the end of the 1st year; but Hess⁴ by the use of the duodenal tube found it present in the new born, although not in large amount, thus confirming the observation of Ibrahim,⁵ who obtained it in a 6-months' fetus. It would appear to increase more rapidly in power than formerly supposed. The peptonizing function (trypsin) is active in the 1st month (Zweifel).⁶ Langendorf⁷ showed its presence in the fetus of 5 months.

Intestine.—The total length of the small intestine in the new born would appear from Vierordt's⁸ statistics to vary considerably, ranging from 2 to 3.5 metres (6.4 to 11.5 feet). The large intestine according to the same authority measures from 0.42 to 0.48 metres (1.38 to 1.57 feet). The relative length of the bowel as compared with that of the body is greater in children than in adults. The small intestine grows rapidly. Beneke⁹ found it at birth $5\frac{3}{4}$ times the body length, at 2 years about $6\frac{1}{2}$ times, at 7 years 5 times, and in adult life but about $4\frac{1}{2}$ times. The large intestine was about equal in length to that of the body, both in the new born and in the adult. The sigmoid flexure is especially long in infants, being at birth, as stated by Treves,¹⁰ almost equal to $\frac{1}{2}$ the total length of the large intestine. It often forms a huge loop running up to the lower border of the liver. It is perhaps partly due to this abnormal length that constipation occurs so frequently in infancy. During the first 4 months the sigmoid diminishes greatly in length, while the rest of the colon proportionately increases, the total length of the large intestine thus remaining unchanged. The vermiform appendix reaches its full length early in life. It is particularly prone to kinking in infancy and childhood, and is abundantly supplied with adenoid tissue, both factors tending to the production of appendicitis. According to the conclusions of Gundobin¹¹ Peyer's patches are well developed, but less numerous in the new born as compared with adults; the number of solitary follicles greater; Lieberkühn's follicles and the villi more abundant; and Brunner's glands numerous but not completely developed. The muscular strength of the intestine is comparatively feeble. Amylolytic ferments are present in the small intestine from birth.

¹ *Jahrb. f. Kinderheilk.*, 1886, XXIV, 377.

² *Daten u. Tabellen*, 1906, 44.

³ *Centralb. f. d. med. Wissenschaft.* 1873, 261.

⁴ *Amer. Jour. Dis. Child.*, 1912, IV, 205.

⁵ *Verhandl. d. Gesellsch. f. Kinderh.*, 1908, XXV, 31.

⁶ *Untersuch. ü. d. Verdaungsapparat. d. Neugeborenen.*, 1874.

⁷ *Arch. f. Anat. u. Physiol.*, 1879, 95.

⁸ *Daten u. Tabellen*, 1906, 117.

⁹ *Deutsch. med. Wochenschr.*, 1880, VI, 433.

¹⁰ *Hunterian Lectures*, 1885, 10.

¹¹ *Jahrb. f. Kinderh.*, 1892, XXXIII, 439.

DIGESTION IN INFANCY

(See also Absorption and Metabolism, p. 48)

The act of sucking has a pump-like quality. The base of the nipple is seized firmly by the infant's lips; the tongue is pressed against the nipple, making a longitudinal gutter in which it may lie, and along which the milk is conducted backward; the soft palate and base of the tongue approach each other and shut off the posterior opening of the oral cavity from the nose; the cheeks sink in slightly, while the lower jaw is depressed, thus producing a partial vacuum, by means of which the milk is drawn from the breast. The sucking cushions already referred to (see Cheeks, p. 34) are supposed to prevent a too great collapse of the cheeks. To nurse satisfactorily the infant must be able to breathe easily through the nose, respiration through it not being interfered with by sucking, although it ceases momentarily during the act of swallowing.

The saliva plays but small part in digestion in early infancy. Later its diastasic action is of service in the case of infants receiving starch. Yet this action is, however, not completed in the mouth, and, after the food is swallowed, should continue only so long as the gastric contents are alkaline or but faintly acid, which is but for a short time.

The stomach is more of a simple receptacle in infancy than is the case later in life. Some of the milk entering the stomach passes immediately through the pylorus (Hess).¹ That remaining is promptly coagulated by the action of the rennin or by the hydrochloric acid present. Tobler² found that the coagulation commenced in 2 or 3 minutes after the milk was ingested. The protein of human milk forms small, loose curds; that of cow's milk a much firmer, larger mass. This may be due to the larger percentage of protein present in cow's milk. The acidity of the gastric contents begins very soon after the ingestion of food, and steadily increases. Van Puteren³ found an acid reaction after 10 minutes, and Leo⁴ after 15 minutes. Yet as the hydrochloric acid as fast as secreted unites with the elements of the milk, it is only toward the end of gastric digestion that free hydrochloric acid can be obtained in small amount, and generally not at all. The degree of acidity in infants is decidedly less than in adult life. Bauer and Deutsch⁵ believed lactic acid to be the predominant one after the ingestion of food during the 1st half-year; and Heiman⁶ found it in half of the breast-fed infants examined. Other investigators, as Sedziūk,⁷ state that lactic acid is seldom found in the stomach of the healthy breast-fed infant. Butyric acid develops in the stomach only in pathological conditions when the amount of hydrochloric acid is diminished. The acidity of the gastric contents seems to depend in large part upon phosphoric acid and its salts.

After the milk has been coagulated its solution by the pepsin begins, the hydrochloric acid now aiding in accomplishing this. Yet much the greater portion of the coagulated milk passes out of the stomach of the infant but little affected in other respects by gastric digestion. There is

¹ Amer. Jour. Dis. Child., 1914, VII, 428.

² Verhandl. der Gesellsch. f. Kinderh., 1906, XXIII, 144.

³ Ueber die Verdaung der Säugekinder in der ersten zwei Lebensmonaten. Arbeiten der Gesellsch. f. Kinderärzte in St. Petersburg, 1889. Ref. Bauer and Deutsch, Jahrb. f. Kinderh., 1898, XLVIII, 27; 68.

⁴ Berl. klin. Wochenschr., 1888, XXV, 981.

⁵ Jahrb. f. Kinderh., 1898, XLVIII, 22.

⁶ Arch. of Ped., 1910, XXVII, 570.

⁷ Przegl. Pedyat., 1913, V, 14. Ref. Arch. f. Kinderh., 1914, LXIII, 271.

no action exerted by the gastric secretion upon the carbohydrate, although the fat is acted upon to some extent by the lipase of the stomach.

The length of time during which the ingested food remains in the infant's stomach probably varies with the infant. Leo¹ found that a considerable portion of the milk had left the stomach within $\frac{1}{2}$ hour, the liquid portion containing the sugar and salts and the uncoagulated milk passing out first, and that in the 1st week of life the organ would sometimes be empty within 1 hour, and in the 1st month generally within $1\frac{1}{2}$ hours. This is confirmed by Clark.² Later a somewhat longer time is required, but the average healthy breast-fed infant retains but little of the food in the stomach longer than at most 2 or $2\frac{1}{2}$ hours. This has been practically confirmed radiologically by Leven and Barret,³ Tobler and Bogen,⁴ Flesch and Péteri,⁵ Ladd,⁶ Pisek and LeWald⁷ and others. The cardiac orifice of the stomach, as proven by the experiments of Cannon⁸ on animals, closes automatically with a certain degree of normal acidity of the gastric contents. The pyloric orifice, on the other hand, as shown by Cowie and Lyon⁹ for infants, opens when a certain degree of acidity is attained. The nature of the food taken influences the time it remains in the stomach. Human milk passes from it more rapidly than cow's milk. The greater the amount of casein, the longer the continuance of the food in the stomach. This fact, previously accepted, was demonstrated radiologically by Ladd.¹⁰ Delay also depends to a large extent upon the amount of fat present (Tobler).¹¹ DeBuys and Henriques¹² demonstrated that the body-posture has much to do with the rapidity of the emptying of the stomach, the rapidity being greatest in the right lateral position. The stomach is of little service in the absorption of food, but a small amount of the sugar and digested protein being taken up here; the fats, water and salts practically not at all.

In the intestine the trypsin of the pancreatic juice accomplishes the peptonizing of the milk not already digested in the stomach; this being probably, as stated, much the larger part. If the contents of the stomach are expelled from it in a hyperacid state no peptonizing by the trypsin takes place, since this ferment requires the presence of an alkaline reaction, and is, in fact, destroyed by acidity of the gastric secretion. Inasmuch as the saliva is in such small amount in early infancy and the diastatic ferment of the pancreas is not secreted to any considerable extent in the 1st month, the power of digesting any starchy food at this period of life would appear to be very limited. It must be stated, however, that investigations by Heubner,¹³ Carstens,¹⁴ Shaw¹⁵ and others apparently prove that even infants of 2 months possess a decided power to digest starch. The

¹ *Loc. cit.*

² Arch. of Ped., 1911, XXVIII, 648.

³ Presse médicale, 1906, XIV, 503.

⁴ Monatsschr. f. Kinderh., 1908, VII, 12.

⁵ Zeit. f. Kinderh., Orig., 1911, II, 263.

⁶ Amer. Jour. Dis. Child., 1913, V, 345.

⁷ Amer. Jour. Dis. Child., 1913, VI, 232.

⁸ Amer. Jour. Physiol., 1908, XXIII, 105.

⁹ Amer. Jour. Dis. Child., 1911, II, 252.

¹⁰ Arch. of Ped., 1913, XXX, 740.

¹¹ Ergebnisse der inn. Med. u. Kinderh., 1908, I, 514.

¹² Amer. Jour. Dis. Child., 1918, XV, 190.

¹³ Berlin. klin. Wochenschr., 1895, 201.

¹⁴ Berlin. klin. Wochenschr., 1895, 1100.

¹⁵ Albany Med. Annals, 1904, XXV, 148.

bile-salts in infancy in combination with the steapsin of the pancreas split the fat, and permit of saponification and the forming of an emulsion. The secretion of the small intestine converts the various sugars into the monosaccharides; galactose, levulose and dextrose.

The peristalsis of infancy is less active than in later childhood or adult life, and the combination of this condition with the unusual relative length of the intestine probably gives the infant an especial ability to digest and absorb the large amount of milk taken. Independently of this, the infant possesses a very active power of absorption. This takes place principally from the small intestine, whence all the elements of the food enter the system, the fat passing in practically only from this region. The large intestine absorbs all; but to a very limited degree.

Bacteria of the Gastro-intestinal Tract.—Mouth.—A considerable number of species of bacteria appear in the mouth even very soon after birth, and with the beginning of the taking of food increases decidedly. With the appearance of the teeth there is a further increase in the number of varieties. Among those oftenest found by Nobécourt and de Vicaris¹ are the *bacillus lactis aërogenes*, *micrococcus candidans*, *bacillus coli*, *micrococcus pyogenes aureus* and *albus*, and the *streptococcus pyogenes* and *salivaris*.

Stomach.—The number of bacteria found normally in the stomach is limited, the hydrochloric acid having destroyed many of those swallowed with the food. Yet there are many contained within the masses of coagulated milk, which the acid cannot affect (Tobler).²

Intestine.—The healthy normal infant exhibits soon after birth a considerable number of species of bacteria in this region. The subject was originally investigated especially by Escherich,³ and in more recent years by Tissier,⁴ Moro⁵ and others. After breast-feeding commences the number of germs increases greatly, although the varieties are not numerous. In general the most prominent bacteria in the intestinal tract in breast-fed infants are the *bacillus bifidus* (Tissier), the *bacillus acidophilus* (Moro), and to a less degree the *bacillus coli* and the *bacillus lactis aërogenes*. There are also sometimes present the *bacillus perfringens*, *bacillus butyricus*, *micrococcus ovalis*, *streptococci*, and certain others in smaller numbers. The upper part of the small intestine exhibits very few germs of any sort. The *bacillus lactis aërogenes* and the *colon bacillus* in small numbers are those chiefly found. The former is more abundant here than in any other portion of the intestinal canal. The lower part of the ileum and the colon contain an abundant growth of bacteria, the *bacillus bifidus* being the predominating germ. There are also present the *bacillus acidophilus*, the *colon bacillus*, the *micrococcus ovalis*, and a diminished number of the *bacillus lactis aërogenes*. The colon and rectum have a smaller number of living bacteria than is found in the cecum.

In artificially fed infants the variety of germs is greatly increased. The *colon bacillus* and intestinal cocci are the predominating micro-organisms, but the others mentioned are also present in large numbers. The *colon bacillus* and the *bacillus lactis aërogenes* are more numerous

¹ Arch. gén. de méd., 1905, CXCVI, 3201.

² Ergebnisse d. inn. Med. u. Kinderh., 1908, I, 495.

³ Darmbakterien des Säuglings, 1886.

⁴ Comptes rend. soc. de biol., 1899, VI, 943. XIII Internat. Med. Cong., 1900. Méd. de l'enf., 208.

⁵ Jahrb. f. Kinderh., 1905, LXI, 687; 870.

than in breast-fed children. Bahrdt and Beifeld¹ emphasize the fact that in the breast-fed infants the germs are especially those producing fermentation; while in the bottle-fed the process is chiefly decomposition. The bacilli of the breast-fed infants tend to be Gram-positive, and those of the artificially fed infants Gram-negative.

Under pathological conditions there may be an enormous increase in the number of the normal bacteria, and various others are present, not native to the intestine; while at the same time germs which are normally present and harmless assume a special virulence and are productive of diseased conditions. Among those found are the *bacillus proteus vulgaris*, *bacillus enteritidis*, *streptococcus enteritidis*, *bacillus pyocyaneus*, and forms of the dysentery bacillus.

Gases of the Digestive Tract.—The gas in the stomach is in part swallowed by the infant while nursing, and in part enters from the intestine. But little is produced by fermentation of the food, and a small quantity appears to be secreted by the gastric mucous membrane. In healthy children it consists of the elements of the atmospheric air only.

The gas in the intestine depends principally on the decomposition of the milk-sugar and consists of CO_2 and H (Escherich).² There are no foul-smelling gases in the intestine of milk-fed infants who are in a healthy condition.

Feces.—The first passages of the infant consist of the meconium. This is a tarry, dark, greenish-brown, almost odorless and faintly acid substance. It is sometimes passed before or during birth, and 3 to 5 times during the first 2 or 3 days of life. It contains cells from the intestine and the skin, minute hairs, fatty granules and globules, cellular detritus, intestinal mucus, and biliary acids, coloring matter, cholesterin crystals and other substances derived from the bile. The source of some of the elements is the amniotic liquid which the fetus has swallowed from time to time. Vierordt³ estimates the total amount of meconium passed as equalling 60 to 90 grams (2.12 to 3.17 oz.). Should the secretion of milk be delayed, the meconium is replaced after 2 or 3 days by stools consisting of brownish or greenish mucus. On the 3d or 4th day, or sometimes earlier or later, the ordinary milk-feces of the infant appear. These are golden-yellow or canary-yellow in color, of salve-like consistence or of that of thin mush, and faintly acid in odor and reaction. When cow's milk is the food employed the stools are alkaline (Biedert),⁴ neutral or faintly acid; of a paler yellow color, have a more unpleasant odor due to the decomposition of the protein, and are often more consistent. Thoroughly digested breast-milk stools appear almost entirely homogenous (Fig. 12). The reaction is, however, according to Schlossmann⁵ not necessarily an indication of the health of the infant, nor is it dependent upon the actual amount of the different ingredients in the food, as much as upon the relationship between them. A ratio of 3 to 1 between the fat and the protein produces an acid stool, while one of 1 to 1 makes the stool alkaline. Consequently the alkaline reaction of the stools of the bottle-fed infant is a natural result of the comparatively high percentage of protein as compared with that of the fat. Very numerous small whitish masses are very common in the stools of

¹ Jahrb. f. Kinderh., 1910, LXXII, Ergänzungsh., 71.

² Darnbakterien des Säuglings, 1886, 160.

³ Gerhardts Handb. d. Kinderkrankh., 1877, I, 118.

⁴ Die Kinderernährung im Säuglingsalter, 1900, 58.

⁵ Centralbl. f. Kinderh., 1906, IX, 237.

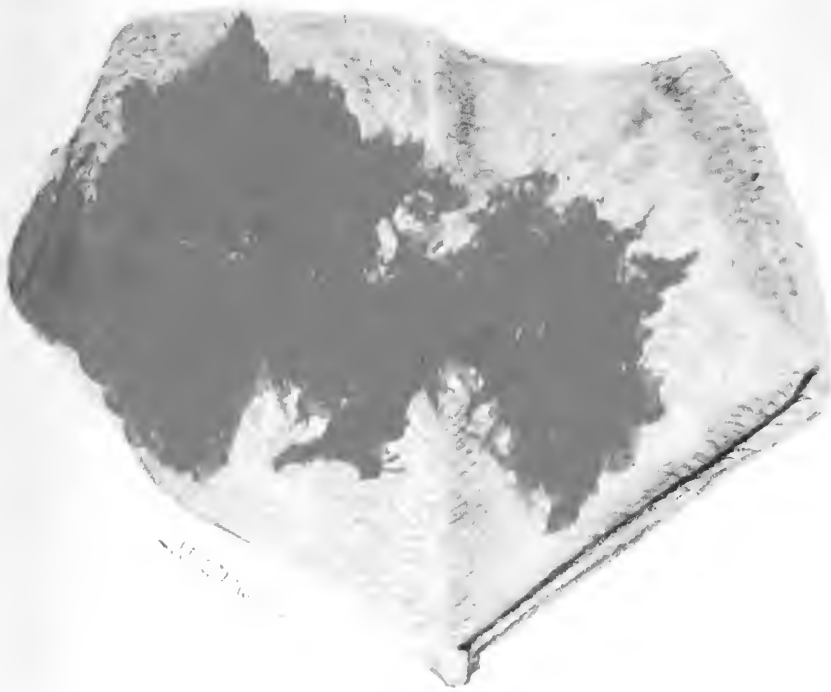
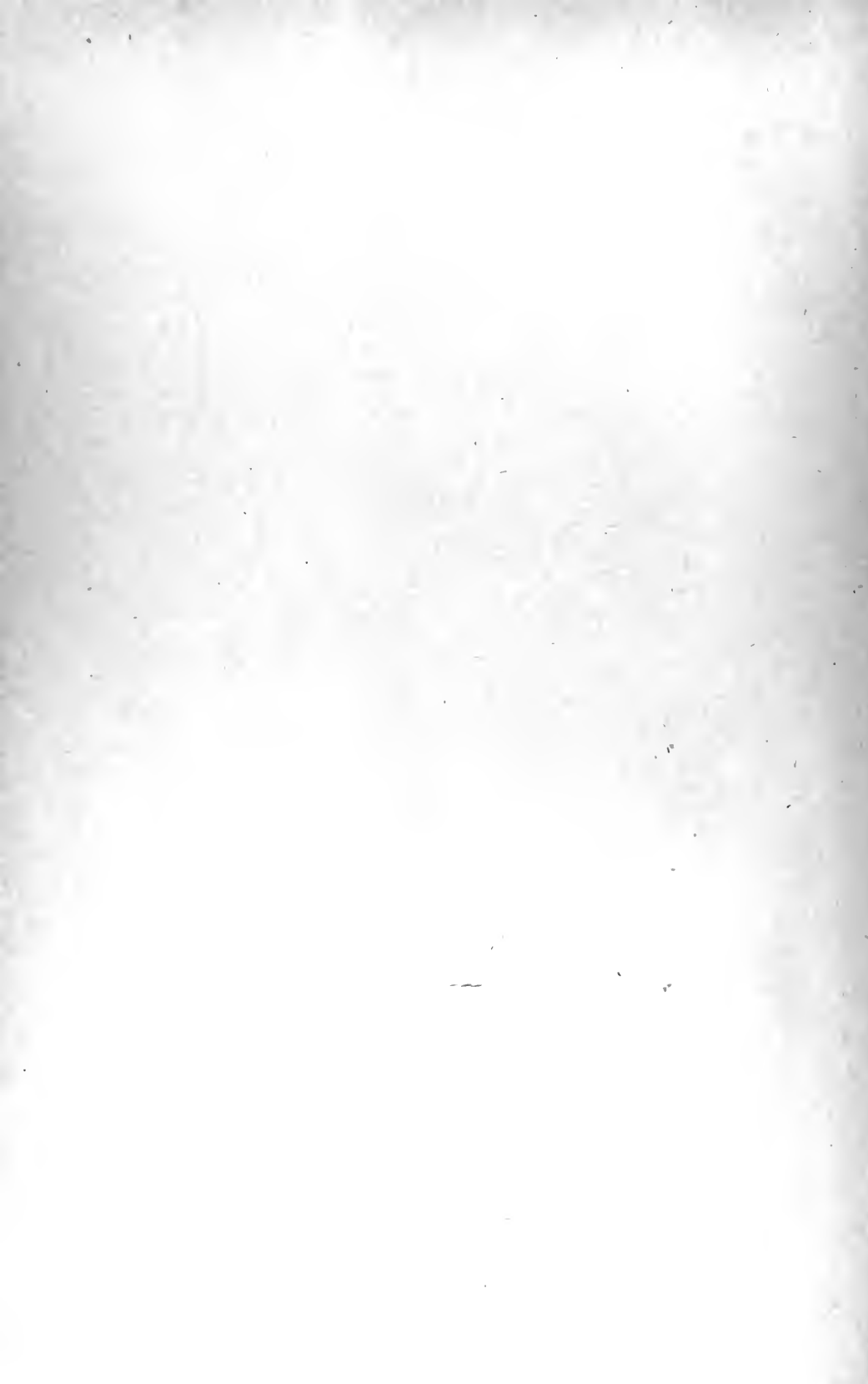


FIG. 12.—THOROUGHLY DIGESTED BREAST-MILK STOOL.



infants apparently entirely healthy and thriving; and this is especially true of those artificially fed. A brownish color may depend upon a relatively large proportion of protein.

The number of intestinal evacuations is at first from 2 to 4 in 24 hours, and after about the 6th week and up to the age of 2 years from 1 to 3. There may be, however, considerable variation from these figures within the bounds of health. The amount of fecal matter passed is estimated by Uffellmann¹ to be about 3 per cent. of the milk taken, or, in the case of feeding with cow's milk, 4.3 per cent.; averaging 3 grams per kilo (21 grains per lb.) of the body-weight, but with a wide range among individual cases.

The stools contain approximately 85 per cent. of water (85.13 per cent. Wegschneider).² The greater part of the residue consists of cellular elements, mucus and bacteria. The milk taken is never wholly absorbed. Fat is always present in the feces, both as neutral fat, in the form of fatty acids, and as soaps from combination of these with the alkalies and alkaline earths. With potassium and sodium an excess of fat forms soft, white curds; with calcium and magnesium insoluble soap-stools are produced. The fat forms from 9 per cent. to 25 per cent. or even more of the dried feces, according to various statistics collected by Biedert.³ The amount may be much in excess of this when the milk is especially rich in this element. The sugar of the food is entirely absorbed. Protein is present only in very small amount in the stools of healthy infants. Knöpfelmacher⁴ found that the greater portion of the nitrogen and phosphorus recovered from the feces of breast-fed infants is derived from the digestive secretions and not from the milk ingested. The protein of this latter has been in part absorbed, in part broken up by the action of bacteria. From 8 to 10 per cent. of the dried feces consists of mineral matter, chiefly calcium, derived partly from the food, partly secreted by the intestine. The proportion of mineral matter is higher in artificially fed infants (Blauberg),⁵ (Heubner).⁶ The small, whitish masses very frequently present in the bowel-movements of healthy, thriving children consist principally of fat or its derivatives and of epithelial cells. The yellow color depends upon the presence of bilirubin which is present unchanged in part. The pale greenish tint so frequently appearing in the stools of healthy children a short time after they are passed, or even present at first, is the result of the oxidation of bilirubin to biliverdin. The acid reaction is due to the lactic acid and the fatty acids present. Such chemical combinations as phenol and skatol, which give the characteristic odor to the stools of adults, are not found in those of milk-fed infants. Various ferments are present in the feces, among them diastase, lactase, invertin, trypsin, rennin, a fat-splitting ferment and others (Hecht).⁷

Under the microscope the bowel-movements of the infant exhibit fat-globules of various sizes; some molecular fat; needles of fatty acids; innumerable bacteria; cholesterine plates; square and columnar epithelial

¹ Deutsch. Arch. f. klin. Med., 1881, XXVIII, 442.

² Ueber die normale Verdauung bei Säuglingen, 1875. Ref. Vierordt, Daten u. Tabellen, 1906, 306.

³ Loc. cit., 61.

⁴ Jahrb. f. Kinderh., 1900, LII, 545.

⁵ Zeit. f. Biol., 1900, XI, 1; 36.

⁶ Verhandl. d. Gesellsch. f. Kinderh., 1901, XVIII, 230.

⁷ Die Faeces des Säuglings und des Kindes, 1910, 148.

cells; small round cells; some thin, granular, yellow, flake-like masses; lime-salts in crystalline form, and occasionally bilirubin crystals, yeast fungi, and proteid matter.

The evacuations become a somewhat darker yellow as the infant grows older, and, when the diet is more varied, and especially when the amount of milk is relatively diminished, they acquire more the characteristics of the stools of adults, both in color and odor. They are still soft, however, as a rule, and acid in reaction. It is only at about the age of 2 years that the stools become formed. This is, however, open to many exceptions, for even young infants may normally pass fully formed stools.

Bacteria of the Feces.—The meconium is at first sterile, but within 24 hours microorganisms enter by the mouth and anus, although not found in large numbers. After breast-feeding begins they are very numerous, but not in great variety. In fact a large part of the stools in infancy is composed of bacteria. Leschziner¹ found that the dried substance of the normal stool of breast-fed infants contains from 2 per cent. to over 28 per cent. of germs; and Strassburger² obtained as much as 42.3 per cent. in normal stools in artificially fed children. In the normal feces of the breast-fed infant are found especially the bacillus bifidus, as well as the bacillus coli, bacillus acidophilus, butyric acid bacillus and sometimes the bacillus lactis aërogenes and others. In the artificially fed infant the bacillus bifidus loses its predominance and is present in association with large numbers of the colon bacillus, bacillus acidophilus, bacillus lactis aërogenes, intestinal cocci, and others, no one type dominating the picture, and the number of varieties being greater than in breast-fed infants. The distinction is to be made between the fermentative and the putrefactive bacteria. Germs of the first class, such as the bacillus bifidus and the bacillus lactis aërogenes, are fermentative and break up milk-sugar into lactic acid and gases, giving an acid reaction to the stool; while the proteolytic bacteria produce, among other actions, decomposition of the protein and give rise to an alkaline reaction. After a mixed diet is commenced there is a further change in the intestinal flora, with an increase in the variety of the microorganisms in the stools.

ABSORPTION AND METABOLISM OF THE FOOD

In addition to and summarizing some of the statements already made under digestion (p. 43) a brief résumé may be given of the physiological processes which attend and follow this, viewed from the point of view of the food-elements rather than the organs and as applicable especially to infancy. A very large amount of investigation in this direction has been made during recent years. For further consideration the reader is referred to the numerous journal articles upon the subject, and especially to the publications of Tobler and Bessau,³ Czerny and Keller,⁴ Langstein and Meyer,⁵ and Morse and Talbot.⁶

Fat.—The amount of fat in the food has a decided influence upon the time this remains in the stomach, as shown by the investigations of Tobler,⁷

¹ Deutsch. Aerzte-Zeitung, 1903, V, 385.

²Zeit. f. klin. Med., 1902, XLVI, 433.

³Brüning u. Schwalbe, Handb. d. allgem. Path. u. d. path. Anat. des Kindesalter, 1912, I, 650.

⁴Des Kindes Ernährung, etc., 1906.

⁵Säuglingsernährung u. Säuglingsstoffwechsel, 1914.

⁶Diseases of Nutrition and Infant Feeding, 1915.

⁷Ergebnisse der inn. Med. u. Kinderh., 1908, I, 514.

the larger the amount of this, the slower being its discharge. This slow entrance into the small intestine permits of a readier digestion of it when it reaches this region. The fat is in no way affected by the saliva, but in the stomach the gastric lipase is able to break up a considerable portion of it. Comparatively little digestion of fat, however, occurs here, and no absorption at all. In the small intestine the fat is split by the pancreatic lipase in combination with the bile-salts and aided by the intestinal secretion, the fatty acids uniting with the alkalies and forming soaps, soluble and insoluble, and later an emulsion. It is in this form that the fat is absorbed by the small intestine, more than 90 per cent. of that ingested both in breast-fed and in normal bottle-fed infants, being utilized. Very little fat is absorbed by the large intestine. It is questionable whether the neutral fat as it occurs in the milk ingested is absorbed at all in this form. There is reason to believe, however, as claimed by Kastle and Loevenhart¹ that the lipase in the intestinal mucous membrane may change the soaps back into neutral fat and that it enters the lacteals in this form. The fat appearing in the stools may possibly be derived in part from the intestinal secretion and from decomposition of the carbohydrates; but probably much the largest portion is from the food. The absorption of fat in artificially fed infants can be modified by altering the proportion of the other elements of the food. Thus in some cases an addition of carbohydrate apparently increases the absorption of the fat and changes a soap-stool to one of a more normal acid character; and, on the other hand, an increase of the protein may similarly change a highly acid, loose stool into a firmer, more alkaline one.

In the economy the principle purpose of the fat is to maintain the body heat. It is an element of the food which it is very difficult to do without for any length of time, for since its caloric value is twice that of either protein or carbohydrate, to replace it an undue amount of one or the other of these must be given.

Carbohydrate.—The only carbohydrate present in milk is lactose; a disaccharide, resolvable into dextrose + galactose (Reuss).² In artificially fed infants the other disaccharides, saccharose (dextrose + levulose) and maltose (dextrose + dextrose), are often employed. Starch, too, a polysaccharide, is to be considered, and in older children cellulose. On the sugar the saliva has no action whatever, nor has the secretion of the pancreas, unless maltose is employed, when there may be a slight reduction by the maltase found present by Ibrahim.³ The starch is converted by the salivary, pancreatic and intestinal secretion into sugar; although some part of it is probably destroyed in infancy by bacterial action within the intestinal canal. The power of digesting starch rapidly increases with an increase of the quantity and strength of the amylolytic ferments. The invertin, maltase and lactase of the intestinal canal act respectively upon the saccharose, maltose and lactose, reducing them to monosaccharides, in which form only are they absorbable under ordinary conditions. Only when given in very large amounts do the disaccharides pass the normal intestinal mucous membrane. They then appear unchanged in the urine, with the exception of maltose, which may be broken up by the maltase of the blood and utilized. The monosaccharides are absorbed rapidly by the small intestine, especially in the upper part, and carried by the portal circulation to the liver where they are changed

¹ Amer. Chem. Jour., 1900, XXIV, 491.

² Wien. med. Wochenschr., 1910, LX, 1635.

³ Verhand. d. Gesellsch. f. Kinderh., 1908, XXV, 32.

into glycogen. The large intestine also possesses to some extent the power of the reduction of the disaccharides and of absorption.

The different sugars are utilized by the infant to different degrees. All of them are fermentable, but lactose undergoes lactic acid fermentation, saccharose more readily alcoholic and less easily butyric acid fermentation, and maltose most easily butyric acid and next readily alcoholic fermentation. Maltose can be assimilated in larger amount than lactose; the latter in about the same quantity as saccharose. Lactose is absorbed more slowly than the others. All in large amount have a tendency to loosen the bowels but lactose to a less degree than maltose. The normal infant can digest from 3.1 to 3.6 grams of lactose or saccharose per kilogram of its body-weight (22 to 25 grains per lb.) and about 7 grams of maltose per kilogram (49 grains per lb.) (Hill).¹

The assimilation, or at least the retention, of the nitrogen of the protein is rendered more complete by the presence of carbohydrates, as shown by Keller,² Orgler³ and others. The carbohydrates would seem, too, when not in excess to favor the absorption of fat, perhaps by preventing the formation of calcium and magnesium soaps. In excess they interfere with the absorption of fat by producing diarrhea, by which the fat and the other intestinal contents are rapidly removed from the body. After absorption the carbohydrates serve to maintain the heat of the body. In addition they have an action in preserving the proper metabolism of the fats, which without carbohydrates are productive of acetone bodies. In the stools the carbohydrates appear chiefly in the form of undigested starch, when this is given in unduly large amount to infants. Very little soluble carbohydrate is found. The gas in the intestine is dependent in part upon the decomposition of the carbohydrate by bacteria. This decomposition also produces the acidity of the stools, which is in direct proportion to the amount of the carbohydrate ingested.

Protein.—The digestion of the protein takes place, as stated, partly in the stomach, under the influence of rennin, pepsin and hydrochloric acid, but chiefly in the intestine by the trypsin of the pancreas and the erepsin of the intestinal secretion. The action of this latter is upon the casein and the peptones and albumoses, changing them into amino-acids, in which form they are absorbed and utilized in the body. It would not appear to be a matter of indifference in what form the protein is present in the food. The value of the large percentage of whey in human milk may depend upon the fact that amino-acids are present in the whey in large amount, but in quite small quantity in the casein. There is almost complete absorption of the protein in normal and normally fed infants, the dried stools showing only from 4 per cent. to 4.5 per cent. to be nitrogen (Orgler),⁴ and even this is chiefly derived from the intestinal secretions and from dead bacteria. The curds in the stools are composed of casein to a very limited extent; the casein curds being hard and tough, and being usually absent in the case of healthy infants. They are less liable to appear when the milk has been boiled (Brennerman).⁵

The needs of the infant in the matter of nitrogen derived from the protein are not great, and the giving of an excess of this is not required. Whether protein is capable of being harmful has been disputed. Certainly

¹ Bost. Med. and Surg. Journ., 1918, CLXXIX, 1.

² Czerny and Keller, *loc. cit.*, 1906, I, 305.

³ Jahrb. f. Kinderh., 1908, LXVII, 390.

⁴ Ref. Thiernich in Feer's Lehrb. d. Kinderh., 1914, 14.

⁵ Amer. Jour. Dis. Child., 1911, I, 341.

it can usually be tolerated in large amounts, and the excess is then utilized in the economy for the production of calories. (See also p. 130.) At least 1.5 grams per kilogram (10.5 grains per lb.) of the body-weight is required daily to maintain a positive nitrogen balance in the system; or in other words 7 per cent. of the caloric requirements should come from the protein. This is approximately equivalent to $1\frac{1}{2}$ oz. of milk for each pound of the weight of the infant (98 c.c. per kilo).

Yet the maintaining of this balance does not depend solely upon the amount ingested. Infants may retain nitrogen even when the number of calories in the food administered is insufficient. While, as stated, the giving of carbohydrate in proper amount favors the retention of nitrogen, the fat has no such favorable action, and in excess may act unfavorably. An increase in the retention of nitrogen is not necessarily attended by a gain in the body-weight. Inasmuch as all proteins must be reduced to amino-acids before they can be absorbed, there would appear to be no difference in the effects produced by them in entirely healthy infants, except, as stated, in the greater amount of amino-acids present in some of them, and the consequent less digestive action required. Yet in spite of the large tolerance for protein shown by most children, there is ample reason to believe that these, especially as seen in the case of the casein of cow's milk given in excess, are capable of producing decided digestive disturbances. This has been emphasized by a number of investigators. The subject is reviewed, among others, by Benjamin¹ and by Talbot and Gamblé.² (See also p. 131.)

Mineral Matter.—The salts of human milk (see p. 97) are readily absorbed from the small intestine, but to a negligible degree from other regions. They may in part re-enter the intestine from the circulation; and in addition salts are contained in the digestive secretions. Consequently, although they are absolutely necessary for digestion and metabolism, their action is complicated and not as yet thoroughly understood. The greater quantity of salts, except iron, in cow's milk as compared with human milk results in the amount taken by the infant artificially fed being always in excess of the needs. In general the absorption and retention of the mineral matter of the food goes hand in hand with that of the nitrogen. Under normal conditions 40 per cent. of the mineral of the ingested cow's milk is lost in the stools. This is chiefly calcium phosphate (Holt, Courtney, and Fales).³ The presence of a sufficient amount of fat in the food increases the retention of the mineral matter (Hoobler).⁴ An excess of it may increase the loss of calcium and of magnesium in the feces by the production of soaps in large amount. Sodium and potassium are absorbed well from the intestine. They are eliminated by the urine and feces. Phosphorus is absorbed better from human milk, since a greater proportion of it is in organic combination than is the case with cow's milk. According to Hoobler⁵ an increase of the fat in the food favors its absorption. The retention of calcium is aided by carbohydrates, unless they are in excess; when the diarrhea resulting causes a decided loss of all mineral matter, although chiefly the potassium and sodium. Calcium may be absorbed either from organic or inorganic combinations, and the part of it which is not retained is excreted through the urine or through

¹ Zeitschr. f. Kinderh., Orig., 1914, X, 185.

² Amer. Jour. Dis. Child., 1916, XII, 333.

³ Amer. Jour. Dis. Child., 1915, IX, 213.

⁴ Amer. Jour. Dis. Child., 1911, II, 107.

⁵ Loc. cit.

the intestinal wall into the feces. The retention of magnesium is, according to Hoobler¹ better when the fat-percentage is low. With all these substances, as with sulphur, the absorption and retention appear to be better in infants receiving human milk.

GASEOUS METABOLISM, ENERGY METABOLISM

An increasingly large amount of investigation has been given to this subject for several years. Most of the results are too technical for inclusion here, and but a brief sketch can be given of some of the data obtained. In the chapter upon Infant Feeding (p. 119) reference will be again made to the direct application of the physiological data to the preparation of the food for an artificially fed infant. The reader is referred to the journal-literature which will be mentioned, and especially to the chapter upon Energy Metabolism in the text-book of Morse and Talbot² for a condensed review of the subject.

Inasmuch as the excretion of carbonic dioxide is, as shown by Rubner and Heubner,³ on the one hand in proportion to the body-surface, and, on the other, an index of the amount of nourishment required, it is evident that the number of calories demanded is directly related to the body-surface of the infant. Since, however, the estimation of the surface is a matter difficult or impossible to compute by the clinician, it has become customary to make the weight of the infant the basis. This can very readily lead to considerable error, inasmuch as it is possible for an infant to lose many pounds of its body-weight, and yet to retain, of course, the same body-surface. Further, it is now doubtful whether even the body-surface is to be considered the guide, but rather the "actual mass of the protoplasmic tissue" (Benedict and Talbot).⁴

In calculating the caloric requirements of the infant it may be reckoned that the basal metabolism of an infant of any age, *i.e.*, the consumption of energy during a state of complete repose after taking food, is equivalent to from 52 to 63 calories per kilogram (24 to 29 calories per pound) of the body-weight. This constitutes the lowest number of calories on which the continuance of health is possible, and only under this condition of inactivity, and does not allow for growth. An exception to this is in new-born infants, as well as in older infants decidedly above the average weight for their age. Here the basal metabolism is usually from 40 to 52 calories per kilogram (18 to 24 calories per pound) of body-weight. Muscular exercise, crying, and the like may increase the energy-requirements occasionally as much as 100 per cent (Benedict and Talbot).⁵ The sick and inactive infant produces less heat than the healthy, lively one. The former may exhibit no greater heat-production than the basal metabolism; the latter decidedly more. The new born requires a minimum of fewer calories probably on account of this inactivity; and the extra-fat children probably partly on this account and partly because their weight is out of proportion to their body-surface. Emaciated infants have a basal metabolism above the figures given, varying from 63 to 87 calories per kilogram (29 to 39 per pound) (Morse and Talbot).

¹ *Loc. cit.*

² Diseases of Nutrition and Infant Feeding, 1915.

³ Zeit. f. Biol., 1898, XXXVI, 1; 1889, XXXVIII, 315.

⁴ Amer. Jour. Dis. Child., 1914, VIII, 1.

⁵ Carnegie Institute Wash., Pub. 201, p. 97. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1915, 58.

This is, however, provided they cry considerably. If very weak and quiet the figures are not so high.

Heubner¹ determined that the average healthy breast-fed infant in its 1st half-year required 100 large calories per kilogram (45 per pound) of body-weight in order to gain properly, the number diminishing gradually in the 2d half-year. This he denominated the "energy-quotient." Premature infants and those under 3 months artificially fed have an energy-quotient of 120 calories (54 per pound). These figures have been largely followed by clinicians in determining the food-requirements of infants. They are, however, far from being universally accepted. Cramer² found that the energy-quotient for the new born was less than 50 calories (23 per pound). Czerny and Keller³ believe Heubner's figures too high, and reported an instance of satisfactory progress on an energy-quotient of 70 (32 per pound); and Ramsey and Alley⁴ came to much the same conclusion; while Ladd⁵ gives from 93 to 159 calories per kilogram (42 to 72 calories per pound) as the range of the energy-quotient in a series of cases studied by him. Other figures could be quoted varying from those of Heubner. The fact appears to be that the energy-quotient can be regarded as no more than an average one and at the best a rough estimate; and it will vary greatly with the individual child, depending largely upon its degree of activity and its age. Heubner's figures may be taken as a guide for the normal child in the 1st half-year; the number of calories required after this gradually decreasing to 70 or 80 per kilogram (30 or 36 per pound) by the end of the 1st year; but children under weight may need from 130 to 150 calories per kilogram (59 to 68 per pound) of body-weight (Morse and Talbot) in the 1st half-year.

Regarding the relationship of the elements of the milk to the caloric needs, although certain numbers of heat-units are produced in the calorimeter by the combustion of these, the figures do not correspond with the "utilizable" calories supplied when the milk is ingested. According to the values given by Rubner⁶ 1 gram of protein ingested produces 4.1 utilizable large calories; 1 gram of fat 9.3 calories, and 1 gram of carbohydrate 4.1 calories. Knowing the percentage of each of these present in the milk-mixture selected and the amount of this taken, it is a simple procedure to calculate the total number of calories received by the infant during the day, and to determine whether the food meets the energy-requirement. There is, it is true, some difference between the energy-production of the different sugars respectively and of starch, but this is small enough to be disregarded. The further adaptation of the knowledge of the caloric value of the food to infant-feeding will be considered in discussing that subject (p. 121).

ORGANS OF RESPIRATION

Upper Respiratory Passages.—The nasal passages in the infant are very narrow, and the sinuses are imperfectly developed. The larynx is situated high in the neck, the lower border of the cricoid being opposite the upper border of the 5th cervical vertebra, instead of opposite the 7th

¹ *Jahrb. f. Kinderh.*, 1910, LXXII, 121. *Lehrb. f. Kinderh.*, 1911, I, 50.

² *Münch. med. Wochenschr.*, 1903, L, 1153.

³ *Des Kindes Ernährung*, etc., 1906, I, 383.

⁴ *Amer. Jour. Dis. Child.*, 1918, XV, 408.

⁵ *Arch. of Ped.*, 1908, XXV, 178.

⁶ *Zeitschr. f. Biol.*, 1885, XXI, 377.

as in adults. By puberty it has descended to the adult position and in boys increases much in size. The space between the vocal cords of the infant is extremely narrow. The bifurcation of the *trachea* is opposite the 3d dorsal vertebra in the new born, but opposite the 4th in adults.

Lungs.—The lungs of the infant at term lie in a collapsed condition at the back of the thorax. After air enters them they are still small in volume as compared with adult life, and continue so throughout childhood. They weigh at birth about 24 grams (0.85 oz.), the relative weight being about the same as in adult life. The lower level during infancy is not quite as low in relationship to the ribs as it is in adult life (Gittings, Fetterolf, and Mitchell).¹

Respiration.—The type of respiration in the infant is generally described as abdominal. The careful experimental studies of Eckerlein² however, show that it is thoracic as well, neither type preponderating constantly, but sometimes one and sometimes the other being evident. The respiration in infancy, and especially in the new born, is very irregular, and the rate is much influenced by the slightest causes. At times quite long pauses take place. This irregularity is perhaps the most striking feature. It is almost constantly present when the child is awake, and may occur even during sleep.

The average rate of respiration in early life can be determined only approximately, and the estimations of investigators differ widely. The following table of the rate at different ages contains average figures only:

TABLE 20.—RATE OF THE RESPIRATION PER MINUTE

New born.....	30 to 50.	Average about 35 to 40.
Balance of 1st year.....	25 to 35.	Average about 30.
1 to 2 years.....	About 28.	
3 to 4 years.....	About 25.	
4 to 15 years.....	20 to 25.	
Adult life.....	16 to 18.	

The rate of respiration is from $\frac{1}{5}$ to $\frac{1}{4}$ less during sleep. Only with the beginning of childhood does the irregularity largely disappear, but even then the rate may be much increased by comparatively slight causes. It is not until the 10th year that the predominating costal type of breathing develops in girls.

ORGANS OF CIRCULATION

Heart. Size.—The heart in the new born weighs about 24 grams (0.85 oz.) according to the statistics of H. Vierordt³ as compared with the weight of between 260 and 300 grams (9.17 and 10.58 oz.) in the adult. It is thus proportionately larger in the infant, equalling 0.76 per cent. of the body-weight in the new born, and 0.46 per cent. in the adult. This disparity is, however, not so marked after the 1st month of life, from which time the heart grows nearly in proportion to the increase in the body-weight, except that at puberty there is a physiological hypertrophy. The right ventricle is comparatively large and strong in early infancy, its walls being almost as thick as those of the left ventricle (v. Starck).⁴ From the 2d year onward, however, the muscle-mass of the right ventricle is not more than one-half that of the left. The foramen ovale—the oval opening between the auricles—exists still at birth. It is situated at the lower posterior por-

¹ Amer. Jour. Dis. Child., 1916, XII, 579.

² Zeitsch. f. Geburtsh. u. Gynäk., 1890, XIX, 120.

³ Arch. f. Anat. u. Path. Suppl. B., 1890, 62.

⁴ Arch. f. Kinderh., 1888, IX, 247.

tion of the auricular septum. From the anterior border of the inferior vena cava arises a thin membrane, the Eustachian valve, which during fetal life diverted the blood from this vessel through the foramen ovale into the left auricle. With the beginning of respiration at birth, and the cessation of circulation in the umbilical vein, blood ceases to pass through this foramen to any extent, and it gradually becomes closed entirely by about the 10th day of life, or often not for some months. (See Vol. II, p. 119.)

Position.—The position of the heart is rather more horizontal in the infant, and regarding its percussion-boundaries the opinions of writers vary considerably. Von Starck's¹ review of these, with his personal observations, make the relative dullness—which is the most important—begin in the 1st year at the 2d left interspace or, oftener, the 2d rib, extend to the right parasternal line, and reach as far as from 1 to 2 cm. (0.4 to 0.8 inch) beyond the left nipple-line. The width at the position of the nipple is 6.6 to 8 cm. (2.6 to 3 inches). At the age of 6 years the highest extent in percussion-dullness is to the 2d intercostal space. It reaches to the right scarcely as far as the parasternal line, and to the left it extends to the nipple-line or slightly beyond it. The greatest average breadth is 10.2 cm. (4.02 inches). At 12 years the relative dullness is bounded by the 3d rib, the right edge of the sternum and the left nipple-line. Its greatest breadth is 11.5 cm. (4.5 inches). The absolute dullness in the 1st year is bounded by the lower edge of the 3d rib, the left border of the sternum and the left nipple-line.

Apex.—The apex-beat in the 1st year is generally found in or oftener beyond the mammillary line, but quite frequently its position cannot be determined. After this period, according to von Starck, although it may lie in various positions with regard to the mammillary line, it is oftenest outside of it up to the 4th year; on the line from this time up to the 7th year, and within it after the 9th year. It is nearly always in the 4th interspace in the 1st year, generally so in the 2d year, in the 4th and 5th interspaces from the 3d to the 6th years, and generally in the 5th interspace after the 7th year. The position of the apex is, however, subject to great variations within physiological limits. A distance of 2 cm. (0.8 inch) outside the nipple line is to be looked upon, however, with suspicion.

Auscultation shows all sounds to be loud, sharp and distinct and more widely diffused than in adults. This is in part owing to the thinness of the chest wall. In infancy and up to the age of 4 or 5 years the second sound is weak over the aortic cartilage, and loudest over the pulmonary cartilage, but is everywhere weaker than the first sound of the heart. These conditions are entirely different from those obtaining in adult life (Hochsinger).²

Blood-vessels.—The blood-vessels in the child are relatively of somewhat greater capacity than in the adult. The pulmonary artery is decidedly larger than the ascending aorta, while in adult life they are of nearly the same size (Beneke).³ The relative size of the heart as compared with the diameter of the ascending aorta is much less in children than in adults (Beneke). As a consequence of these two relations the general arterial tension is less than in adult life (see p. 58), and the blood-pressure in the lungs in childhood is greater than that of the general arterial system.

¹ *Loc. cit.*, 241.

² Die Auscultation des kindlichen Herzens, 1890.

³ Constitution u. constit. Kranksein, 1881. Ref. Vierordt, Daten u. Tabellen, 1906, 171.

At the point where the pulmonary artery divides into its two main branches springs in the fetus the ductus arteriosus Botalli. Although the largest branch, this vessel is but a short trunk about $\frac{1}{2}$ inch (1.3 cm.) in length at birth. It passes obliquely upward and joins the aorta just below the origin of the left subclavian artery. Since with the beginning of respiration the blood is diverted from the ductus arteriosus into the lungs, this canal closes in from 1 to 2 weeks, persisting only as a small fibrous cord in later life. Among other vessels characteristic of antenatal life, and still present at birth, are the hypogastric, and their continuation the umbilical, arteries. These are stout trunks arising from the internal iliac arteries and passing upward beside the bladder to and through the navel, whence, twisted around the umbilical vein, they reach the placenta. The distal portions of the arteries within the body of the child close completely in from 2 to 5 days after birth, forming fibrous bands, the anterior ligaments of the bladder. The proximal portions remain previous for only a short distance from their origin as the superior vesical arteries. The umbilical vein after entering the body from the navel passes upward along the free margin of the suspensory ligament of the liver. After giving off small branches to the hepatic substance it divides in the transverse fissure into two main branches, of which the larger and shorter joins the portal vein. The other, the ductus venosus, continues along the posterior longitudinal fissure of the liver and joins the hepatic vein where this empties into the ascending cava. Both the umbilical vein and the ductus venosus close completely in from 2 to 5 days after birth, the part of the former within the body finally becoming the round ligament of the liver.

The activity of the circulation is greater in the child. K. Vierordt¹ estimates that the time elapsing from the moment the blood leaves the heart until it returns to it is in the new born 12.1 seconds, at 3 years 15.0 seconds, at 14 years 18.6 seconds and in the adult 22.1 seconds.

The circulation during fetal life and the changes which take place at birth have so intimate a relation to congenital diseases of the heart that they will be described in connection with cardiac disorders.

Pulse.—Even in health the pulse tends to be somewhat irregular in force and frequency, especially during infancy, and even later than this a certain degree of irregularity is common and cannot be considered pathological. Trifling causes, such as crying, nursing or any excitement or exercise increase the pulse rate from 20 to 40 beats per minute. It is often not easy to feel the radial pulse in the first months. The rate is less when the child is lying than when sitting, and sitting than when standing, and is 16 to 20 beats less during sleep. It diminishes also with increasing age, but the figures of different observers vary much. An approximation would be expressed by the following:

TABLE 21.—PULSE-RATE DURING INFANCY AND CHILDHOOD

Birth.....	130 to 150
1st month.....	120 to 140
1 to 6 months.....	About 130
6 to 8 months.....	About 120
1 to 2 years.....	110 to 120
2 to 4 years.....	90 to 110
6 to 10 years.....	90 to 100
10 to 14 years.....	80 to 90

The rate is slightly greater in females except in the first few months.

¹ Gerhard's Handb. d. Kinderkrh., I, 107.

TABLE 22.—BLOOD-PRESSURE IN NORMAL CHILDREN

Age	L. Gordon ¹		Stowell ²		M. Leitão ³		W. Kaupet		W. Kriss ⁵		A. Katzenberger		Judson and Nicholson ⁷	
	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.	S.	D.
3 years.....	81	..	91	..	65	..	90	81.8	80	74	91.8	65.6
4 years.....	83	..	89	..	71	..	86.4	78.3	83	76	91.6	64.9
5 years.....	86.5	..	95	..	71	..	82.2	74	83	76	91.3	64.4
6 years.....	88.5	..	96	97.5	82.1	90	82	92.6	67.3
7 years.....	85	..	102	91	78	90	82	94	66.3
8 years.....	93	..	101	90	88	93.6	64.7
9 years.....	100	..	102	90.5	80.2	90	88	93.6	64.7
10 years.....	95	..	112	90	81.5	90	88	94.3	71
11 years.....	104	..	102	97.7	82.6	98	90	99.2	67.1
12 years.....	105	..	111	96	87.2	98	90	97.1	65.5
13 years.....	107	92	83.1	99	95	102.3	65.2
14 years.....	110	96.6	88	99	95	103.6	70.5
15 years.....	109	107	96	101	96	106.1	67.4
							101	96	105.6	67.5

¹ Arch. of Ped., 1911, XXVIII, 343.² Arch. of Ped., 1908, XXV, 91.³ Arch. d. méd. des enf., 1913, XVI, 102.⁴ Montschr. f. Kinderh., 1910-11, Orig., IX, 257.⁵ Arch. f. Kinderh., 1910, LIII, 332.⁶ Ztschr. f. Kinderh., Orig., 1913, IX, 167.⁷ Amer. Jour. Dis. Child., 1914, VIII, 257.

Townsend¹ has shown that at birth sex has no influence on the rapidity. Dichrotism is absent or imperfectly developed in the pulse of children, and does not appear until the age of from 10 to 14 years (Keating and Edwards).²

Blood-pressure.—The blood-pressure is lowest in children, and exhibits little if any increase until puberty is approached, when a more abrupt increase occurs. Probably the most extensive and carefully conducted studies are those by Judson and Nicholson,³ and their results may be taken as the most reliable. These are shown in comparison with the investigations of others in the table rearranged from that given by them. The author⁴ found it impossible to determine any reliable average normal readings for subjects under 3 years of age. The figures in the table indicate measures recorded in millimeters of mercury.

BLOOD

Blood. Amount and Specific Gravity.—The blood in the new born constitutes about $\frac{1}{15}$ of the body-weight against $\frac{1}{13}$ in adult life. If tying of the cord is deferred the percentage is temporarily increased ($\frac{1}{6}$ of the body-weight (Schücking).⁵ The specific gravity in the new born is estimated by Lloyd Jones⁶ to be highest at birth, equalling 1066, but by the end of the 2d week it has fallen rapidly and continues to decrease until the age of 2 or 3 years when it equals 1048 or 1050. Then it gradually increases until puberty.

Hemoglobin.—The percentage of hemoglobin is generally conceded to be higher in the new born, being then decidedly over 100 per cent. It diminishes rapidly and reaches its minimum in the 1st month, falling almost to 50 per cent. This low percentage continues for the first 6 months or 1st year, after which it rises slowly, keeping pace with the specific gravity. The percentage in childhood varies normally from 65 to 95 per cent.

Erythrocytes (see also Vol. II, p. 454 and Fig. 394).—The number of red blood-corpuscles varies greatly according to different statistics. It is relatively high in the new born, exceeding the proportion in adult life. Stengel and White⁷ give 5,742,080 per c.mm. as the average at birth obtained from the statistics of a number of observers. Biffi and Galli⁸ found the number as high as 7,000,000 in one instance. A decrease in number begins after the 2d day and goes on rapidly, about 500,000 being lost in the first 2 weeks (Schiff).⁹ The diminution continues to exist during the 1st year, and then there follows a gradual increase up to the age of from 8 to 12 years when the number normal for adults is attained (Stengel and White). The average number of red blood-cells in early and later childhood is 4,000,000 to 4,500,000. Nucleated red blood-corpuscles (normoblasts) occur in the fetus, and in small numbers during the early days of life, but after the 1st week their presence is unusual. The size of the red corpuscles varies very much in the new born, pale corpuscles deprived of

¹ Bost. Med. and Surg. Jour., 1896, CXXXIV, 484.

² Arch. of Ped., 1888, Dec.

³ Amer. Jour. Dis. Child., 1914, VIII, 257.

⁴ Personal communication.

⁵ Berl. klin. Wochenschr., 1879, XVI, 582.

⁶ Journal of Physiol., 1887, VIII, 19; 1891, XII, 299.

⁷ University of Pennsylvania Medical Bulletin, 1901, November.

⁸ Journ. de physiol. et de pathol. générale, 1907, IX, 721.

⁹ Zeit. f. Heilk., 1890, XI, 17.

their hemoglobin (shadow-corpuscles) are present in considerable numbers (Silbermann)¹ and poikilocytes may be seen. The fragility of the red cells appears to be greater than in adult life. The coagulability does not differ materially from that of the blood of the adult. Flusser² found the average coagulation-time in the new born $8\frac{1}{4}$ minutes.

Leucocytes (see also Vol. II, p. 455 and Fig. 394).—The leucocytes are present in relatively large numbers in the new born, equalling 16,000 to 20,000 or more to the c.mm. in the 1st days of life, but diminishing to 12,000 or 13,000 by the age of 2 weeks (Schiff).³ There is little change from these figures during the 1st year of life (Gundobin),⁴ but after this period the number steadily falls, averaging according to Karnizki⁵ approximately 9500 at from 1 to 6 years, and 8000 at from 6 to 15 years. Rabinowitz,⁶ however, found the total average number of leucocytes in the first 15 years from 6000 to 7000 as in adults. There may be a very considerable variation within physiological limits. Perlin⁷ gives approximately 8000 to 13,000 as the number at from 4 to 8 years of age, and 7000 to 9000 at 16 years of age. The proportion among the different forms of leucocytes is very variable in infancy and childhood. At birth the polymorphonuclear neutrophiles constitute nearly 75 per cent. (Carstanjen)⁸ but within 48 hours the lymphocytes begin to increase in number. Gundobin gives the proportion of lymphocytes and allied forms in infancy as 50 to 60 per cent. and that of polymorphonuclear neutrophiles as 28 to 40 per cent., and these figures may be assumed as approximately correct. In adults they are 24 to 38 per cent. and 62 to 70 per cent. respectively. These proportions alter gradually, and by the age of 3 years they approach to, and by the age of 8 or 10 years do not differ materially from those of adult life. The number of eosinophiles is much more variable in the blood of infants and children, and may be considerably increased even up to 6 per cent. (Carstanjen) without having any pathological significance. In the first weeks of life a few myelocytes may be found and even after this period they may occur in a large variety of pathological conditions (Zelenski and Cybulski).⁹ A description of the different varieties of red and of white cells and their significance will be given under Diseases of the Blood (Vol. II, p. 454).

GENITO-URINARY SYSTEM

Organs.—The kidneys are distinctly lobulated and comparatively large at birth and generally extend lower than the crest of the ilium especially upon the right side. Their weight at birth is estimated by H. Vierordt¹⁰ at about 23 grams (0.81 oz.) or 0.75 per cent. of the total body-weight, against 0.46 per cent. of the body-weight in adult life. A few years after birth the kidneys assume the position occupied in later life. The renal structure in the new born often exhibits on section reddish yellow streaks toward the apices of the papillæ. These consist of deposits of urates in the tubules, the uric acid infarcts described by Virchow.

¹ Jahrb. f. Kinderh., 1887, XXVI, 252.

² Monatssehr. f. Kinderh., Orig., 1914, XII, 705.

³ *Loc. cit.*

⁴ Jahrb. f. Kinderh., 1893, XXXV, 191.

⁵ Arch. f. Kinderh., 1903, XXXVI, 42.

⁶ Arch. f. Kinderh., 1912, LIX, 101.

⁷ Jahrb. f. Kinderh., 1903, LVIII, 549.

⁸ Jahrb. f. Kinderh., 1900, LII, 215.

⁹ Jahrb. f. Kinderh., 1904, LX, 884.

¹⁰ Arch. f. Anat. u. Phys.; Anat. Abth.; Suppl. Band, 1890.

The **bladder** in the infant is practically an abdominal organ, as the small pelvis is not capable of containing it. The **prepuce** has normally an extremely narrow orifice and is often more or less adherent to the glans. The cervix of the **uterus** is relatively long and the body small. The organ grows but little until puberty. It is more antverted than in adult life. The **ovaries** lie in the abdominal cavity at birth and are relatively large (0.8 grams (0.03 oz.)). (Adult 7.5 grams (0.25 oz.) H. Vierordt).¹ The **testicles** are comparatively small (0.8 grams (0.03 oz.)). They are generally found in the scrotum at birth. The **mammary glands** very frequently become somewhat swollen and begin to secrete a milk-like fluid when the infant is 3 or 4 days old. This secretion is most abundant about the 10th day of life, and may continue for several weeks. This condition of the breasts may be considered physiological. It occurs in either sex equally well, and is independent of the general state of health. The fluid, sometimes called "witch's milk," chiefly resembles in appearance and composition the colostrum secreted by the mother before the birth of the child (Genser).² Colostrum corpuscles are always present. The amount secreted is generally very small, unless the gland has been repeatedly irritated, as by pressure, when even as much as 1 or 2 c.c. (16 to 32 minims) may be obtained at a time.

Urine. Amount.—Urine is, as a rule, present in the bladder at birth, but it is in small quantity, and but little is secreted during the first 2 or 3 days of life. The average amount at the first passage is 9.6 c.c. (0.326 fl.oz.), according to the observation of Martin and Ruge.³ The total daily quantity secreted in the first 2 days is inconstant, but averages much less than later. As soon as the child begins to obtain milk from the mother the secretion of urine is much increased, and continues throughout childhood to be proportionately greater than in adult life. The amount is exceedingly variable, and the statements of different investigators differ widely. It is influenced by many causes, among them the amount of liquid ingested, the temperature of the air, and the state of the digestion or of the nervous system.

The following figures, based upon many estimations, are fairly representative approximations:

TABLE 23.—THE DAILY SECRETION OF URINE

1st and 2d days.....	15-60	c.c. (0.5- 2.0 fl.oz.)
3d to 10th day.....	100-300	c.c. (3.4-10.1 fl.oz.)
10th day to 2 months.....	250-450	c.c. (8.5-15.2 fl.oz.)
2 months to 1 year.....	400-500	c.c. (13.5-16.9 fl.oz.)
1 to 3 years.....	500-600	c.c. (16.9-20.3 fl.oz.)
3 to 5 years.....	600-700	c.c. (20.3-23.7 fl.oz.)
5 to 8 years.....	650-1000	c.c. (22.0-33.8 fl.oz.)
8 to 14 years.....	800-1400	c.c. (27.0-47.3 fl.oz.)

The studies of Churchill⁴ give amounts decidedly less than these. Fuller details may be found in the writings of Martin and Ruge,⁵ Cruse,⁶ Herz,⁷ Schiff,⁸ Camerer,⁹ Lesne and Merklen¹⁰ and others.

¹ Daten u. Tabellen, 1890, 29.

² Jahrb. f. Kinderh., 1876, IX, 160.

³ Zeitsch. f. Geburtsh. u. Frauenkr., 1876, I, 279.

⁴ Arch. of Ped., 1898, XV, 646.

⁵ Loc. cit.

⁶ Jahrb. f. Kinderh., 1877, XI, 393.

⁷ Wien. med. Wochenschr., 1888, XXXVIII, 1510.

⁸ Jahrb. f. Kinderh., 1893, XXXV, 21.

⁹ Wurtenb. Correspondbl., 1876, XLVI, No. 11.

¹⁰ Rev. mens. des mal. de l'enf., 1901, 61.

Frequency of Micturition.—This varies from 2 or 3 up to 6 times on the 1st and 2d days of life. Quite commonly the evacuation does not take place until more than 12 hours from birth, and not infrequently not until on the 2d or even the 3d day of life. After this excretion is very frequent during infancy, varying anywhere from 5 or 6 to even sometimes 30 or 40 times in the twenty-four hours, the urine being often retained several hours during sleep. Engel¹ studied the frequency of micturition by an automatically registering electrical apparatus, and found it varying from 10 to 30 times in twenty-four hours, with a normal average of 25. After control of the bladder is obtained the frequency of urination varies from 6 to 8 times in twenty-four hours.

Physical and Chemical Characteristics of the Urine.—The *specific gravity* of the urine during the first few days of life is high as compared with that of later periods (1012, Martin and Ruge).² After the ingestion of milk begins it rapidly falls to 1002–1006, but when a mixed diet is commenced it gradually increases, and when the child is 5 or 6 years old the specific gravity is about the same as in the adult.

In *appearance* the secretion is at first highly colored and slightly turbid, owing to the concentration and to the presence of urates and mucus. Later, even during childhood, it is generally of a paler yellow than in adult life. Sometimes in infancy, particularly in the new born, it stains the diaper a faintly reddish color through the deposition of urates. The *reaction*, at first decidedly acid, soon becomes usually neutral. *Odor* is almost absent in infancy and even in childhood, unless the urine is high-colored. The ammoniacal odor often noted in the nursery usually is due to lack of care in changing the diapers, the urine decomposing after it has been passed. This is especially true if indigestion is present.

As regards the *chemical constituents* there is very little or no urea in the urine at birth. The proportion is much increased by the 3d day, but is still relatively low during infancy. Phosphates, chlorides and sulphates are also present in relatively small amounts. The proportions of all of these are increased when a mixed diet is commenced, but are still less than in adults. The amount of urea, however, as compared with the body weight is greater in childhood than in adult life. The percentage of uric acid is especially large in the new born, and, though then diminishing, still remains throughout childhood in excess of that of adult life. The relation of uric acid to urea is 1:14 in the new born, and but about 1:70 in the adult.

The urine of healthy breast-fed infants usually contains no indican, but in those fed artificially it is generally present in small quantity. Older children on mixed diet exhibit indican to the same extent as do adults.

Albumin in small amount may very often be found in the urine of healthy new-born infants. Traces of sugar may sometimes occur in healthy infants, according to the statements of a number of investigators. Reuss³ found glyocol a normal constituent in the new born, and Ostrowski⁴ observed urobilinuria frequently in healthy infants. Rennin and pepsin are said by Pechstein⁵ to be always discoverable in the urine of children. Small amounts of the acetone-bodies are normally present

¹ Deut. med. Wochenschr., 1914, XL, 1960.

² Loc. cit.

³ Zeit. f. Kinderh., Orig., 1911–12, III, 12; 286.

⁴ Przegląd lekarski, 1912, No. 10. Ref. Monatsschr. f. Kinderh., Ref., 1913, XII, 172.

⁵ Zeit. f. Kinderh., Orig., 1911, I, 357.

(Veeder and Johnston);¹ and phenol is constantly found (Moore).² *Microscopically* nothing characteristic is noticed except that the presence of hyaline casts is not unusual in the case of young infants.

THYMUS GLAND

The thymus gland is essentially an organ of early life. Its size is subject to great variation. According to Friedleben,³ whose figures have been much quoted, the length from birth to the 9th month averages approximately 6.9 cm. (2.71 inches); from 9 months to puberty 8.4 cm. (3.30 inches), and in adult life from 10 to 13 cm. (3.94 to 5.12 inches), the glandular tissue being then largely replaced by fat. At birth the gland fills up much of the space in the lower anterior portion of the neck and behind the upper part of the sternum. Its weight, according to his figures,⁴ are:

TABLE 24

Birth.....	14.3 grams (0.51 oz.)
1-9 months.....	20.7 grams (0.73 oz.)
9-24 months.....	27.3 grams (0.96 oz.)
2-14 years.....	27.0 grams (0.95 oz.)
15-25 years.....	22.1 grams (0.78 oz.)
25-35 years.....	3.1 grams (0.11 oz.)

Some more recent investigations gave reason to believe, however, that these estimations are too high. The careful studies of Bovaird and Nicoll,⁵ based upon the examination of 495 glands, furnish figures for weight decidedly less. The birth-weight was found to average 7.7 grams (0.27 oz.), with a decrease to 5.9 grams (0.21 oz.) for the first 5 years of life, and a still further diminution after this period. Dudgeon⁶ found the normal weight in infants up to 2 years of age to be from 7 to 10 grams (0.25 to 0.35 oz.). Vierordt,⁷ from his comparison of different statistics, placed the average birth-weight as 8.15 grams (0.29 oz.). On the other hand, Hammar,⁸ in a study upon the thymus glands of 126 well-nourished individuals dying suddenly or after very acute illness, and consequently with presumably normal glands, found that the weight at birth averaged 13.26 grams (0.47 oz.); from 1 to 5 years 22.98 grams (0.81 oz.); from 6 to 10 years 26.10 grams (0.92 oz.), and from 11 to 15 years 37.52 grams (1.32 oz.). It is evident that a decided difference of opinion still exists which has yet to be settled; but it would seem safe to assume that any gland weighing more than 10 to 15 grams (0.35 to 0.53 oz.) is above the average weight, although the possible range in normal weights is to be borne in mind (from 5 to 20 grams) (0.18 to 0.70 oz.) (Schridde).⁹ The gland is loosely attached to the sternum, but firmly to the pericardium. Its position may be recognized by radiography as well as by percussion. (See Diseases of the Thymus Gland, Vol. II, p. 517.)

¹ Amer. Jour. Dis. Child., 1916, XI, 291.

² Amer. Jour. Dis. Child., 1917, XIII, 15.

³ Die Physiologie der Thymusdrüse, 1858.

⁴ As Friedleben's figures are given in the old grains of Cologne, I have used the equivalents as quoted by Friedjung (Pfaundler und Schlossmann, Handb. der Kinderh., 1906, II, 1, 394).

⁵ Arch. of Ped., 1906, XXIII, 641.

⁶ Path. Soc. Transac., London, 1904, LV, 151.

⁷ Daten und Tabellen, 1906, 44.

⁸ Ergebn. d. Anat. u. Entwicklungesch., 1909, XIX, 253.

⁹ Münch. med. Woch., 1914, LXI, 2161.

SUPRARENAL BODIES

The suprarenal bodies are relatively very large in infancy, weighing about as much as in adult life.

SPLEEN

The spleen weighs approximately 10 grams (0.35 oz.) at birth (Vierordt).¹ Its relative weight is slightly greater at this period than in adult life. (Adult 163 grams (5.75 oz.) Vierordt.)

THYROID GLAND

The thyroid gland is comparatively large in infancy, weighing, according to the statistics of Vierordt,² 4.85 to 9.75 grams (0.17 to 0.34 oz.). This makes its size as compared with the body-weight 3 times as great as in adult life. The isthmus is small in children, but can be discovered by palpation, if the subcutaneous fatty tissue of the neck is not too abundant at the point where it crosses the trachea below the cricoid cartilage. The lateral lobes, however, cannot be felt, and even a somewhat enlarged gland may be impossible of recognition during life.

TEMPERATURE

The temperature of the body at birth, taken in the rectum, averages in the neighborhood of 37.8°C. (100.4°F.) (Vierordt).³ Within an hour or two it falls temporarily about 1.7°C. (3.1°F.) and then, before or by the end of the 1st day of life, rises again to 37.6°C. (99.7°F.) (37.59°C. Förster).⁴ Throughout infancy a daily rise in temperature begins at 2 or 3 A. M., which gradually increases and reaches its maximum in the early afternoon, to be followed by a fall which commences toward evening and continues until after midnight. The very extensive investigations of Jundell,⁵ upon over 3000 records, show that the daily fluctuation in the second half of the 1st week in perfectly healthy infants does not amount to more than 0.1°C. (0.18°F.), by the age of 1 month averages about 0.25°C. (0.45°F.), and by 6 months reaches a range of 0.5°C. (0.9°F.). In early childhood the daily fluctuation amounts to nearly 1°C. (1.8°F.), which is somewhat greater than in adult life. The average temperature of childhood is rather higher than in adults (0.3°C. Vierordt) (0.54°F.), and elevations are more readily produced by slighter causes. After the ingestion of nourishment infants show a slight fall of temperature, followed soon by a slight temporary rise above that existing before nursing (Demme).⁶ The temperature during sleep is slightly lower than when the infant is awake, and somewhat higher after exercise or crying. External influences, too, affect it. Thus variations in the temperature of the surroundings may elevate or depress that of the infant. It is to be noted also that the axillary temperature in normal children is from 0.3 to 0.9°C. (0.54° to 1.6°F.) less than the rectal (Demme), and in sick children from 0.5 to 1.1°C. (0.9 to 2°F.).

¹ Daten u. Tabellen, 1906, 29.

² Daten u. Tabellen, 1906, 42.

³ Daten u. Tabellen, 1906, 360.

⁴ Journ. f. Kinderkrh., 1862, XXXIX, 1.

⁵ Jahrb. f. Kinderh., 1904, LIX, 521.

⁶ 14. med. Bericht. d. Jennersch. Kindersp. in Bern, 1877, 7. Ref. Vierordt, *loc. cit.*, 364.

NERVOUS SYSTEM

Brain.—The brain of the new born is proportionately very heavy, equalling about 380 grams (13.4 oz.) or from 12 to 13 per cent. of the body-weight, while in adults it is only about 2 per cent. Growth is rapid, especially in the 1st year, the weight increasing nearly $2\frac{1}{2}$ times. After about the 5th year, however, the rate of increase in weight is very slow.

The following table, after Vierordt,¹ shows the weight at different ages:

TABLE 25.—WEIGHT OF THE BRAIN AT DIFFERENT AGES

	Male	Female
Birth.....	381 grams (13.4 oz.)	384 grams (13.5 oz.)
6 months.....	632 grams (22.3 oz.)	575 grams (20.3 oz.)
1 year.....	945 grams (33.3 oz.)	872 grams (30.8 oz.)
2 years.....	1025 grams (36.2 oz.)	961 grams (34.0 oz.)
5 years.....	1263 grams (44.6 oz.)	1221 grams (43.1 oz.)
10 years.....	1408 grams (49.7 oz.)	1284 grams (45.3 oz.)
25 years.....	1431 grams (50.5 oz.)	1224 grams (43.2 oz.)

The cerebellum is relatively smaller than the cerebrum as compared with adult life. The brain-substance is very soft at birth, and the grey matter is not sharply differentiated from the white. Although the convolutions are less evident than in adult life they all become visible by the age of 5 weeks. The dura mater is adherent during the 1st and often also during the 2d year. There is a greater amount of fluid in the subdural space than later in life.

Spinal Cord.—The spinal cord at birth is comparatively heavy, weighing 5.5 grams (0.19 oz.) or 0.18 per cent. of the body-weight (Vierordt) against 0.06 per cent. in adults. At birth it extends downward sometimes to the 3d lumbar vertebra, but in other cases only to the 1st, as in adults.

DEVELOPMENT OF MUSCULAR AND NERVOUS FUNCTIONS

For the first few weeks of life the infant lies very still wherever placed, unable to change its position, and sleeping most of the twenty-four hours. The action of the flexor muscles preponderates to some extent, and the hands are usually clinched much of the time, the head sunken forward, the back convex, and the forearms, thighs and legs flexed and drawn to the body. The head cannot be held erect, the alterations in the expression of the face are meaningless, and the apparent smile sometimes seen is not expressive of comfort. Any movements which occur are automatic or reflex. Sucking at the nipple, for instance, and the grasping by the hand of an object placed in it are done purely unconsciously. This automatic grasp of the new born is very powerful; so much so that the child can sometimes be raised entirely from the bed before it relaxes. Soon a very distinct increase in general power takes place. The motions of the legs and arms become very active, but uncontrolled and still purposeless. In the 2d month the head can be held upright to some extent, and by the 3d or 4th month very well. By the age of 3 months, or sometimes a little sooner, purposeful efforts at grasping objects begin, but without any idea whatever of distance being shown. By 6 months, although the

¹ Arch. f. Anat. u. Phys.; Anat. Abth.; Suppl. Band, 1890, 62.

motions are still largely impulsive, the child can make many well-directed movements and can grasp for and play with its toys. At this age it can sit supported very well, and unsupported to some extent, although frequently falling backward until the age of 9 or 10 months. The age when the infant is able to roll over varies greatly. A few can accomplish this by 3 months, but the majority not until much later.

At about the age of 6 months the infant will often try to stand if held on its feet in the lap. At 7 or 8 months it makes attempts at creeping or at moving along the floor or bed in some other manner. Some children, however, never creep. When a year old, or sometimes even when 9 or 10 months, it will stand, holding to objects. Walking, while supporting itself by the wall or by furniture begins soon after 1 year of age, the time varying greatly, and the power to walk without support is gained in a few months more. Falls are, of course, very frequent, and these are nearly always backward, bringing the child into a sitting position. This is due to the comparative weakness of the extensor muscles. The toes are always turned in when walking, and this condition is overcome only very gradually.

The time for the acquisition of muscular and nervous control of the passage of urine depends largely upon training. With care it is sometimes possible to teach an infant of even 3 or 4 months, at least during the day. Usually, however, the control by day is not acquired until sometime in the 2d year. The age of 2 years is an extreme limit for children with whom any effort at instruction has been made. The same statements apply to the control of the fecal evacuations.

Reflex Action.—This is, for the most part, well-developed in the new born. Many of its forms are entirely uncontrolled by the inhibitory influence which develops later; as, for instance, reflex evacuation of the bowels and the bladder. Some of the reflex movements which persist throughout life, as the plantar and the patellar reflexes, are, on the whole, not so uniformly well shown in the 1st and 2d years as later. Others, such as the abdominal, cremasteric, corneal and pupillary reflexes, are fully developed from birth. The investigations of Engstler¹ and of Levi² showed that the plantar reflex in the new born is characterized by *dorsal* flexion of the toes (Babinski reflex). Gradually this condition changes, but it is not until the 3d year that plantar flexion is the rule.

DEVELOPMENT OF SPECIAL SENSES AND MENTAL POWERS

Sight.—In the first weeks the child probably cannot see, except to distinguish light from darkness, and will not wink when the finger is brought near the eye. The perception of light is decided, and sometimes evidently unpleasant, since the infant closes its lids whenever the light is too bright. The eyes are expressionless and move slowly, and more or less of lack of coordination persists until the age of 3 months. The pupils react promptly to light at once after birth. Between the ages of 3 and 6 weeks the baby can fix its eyes upon objects, but even by the 6th day it may turn its face to the light. By 7 weeks the reflex closing of the lids on the approach of an object close to the eyes is well developed. Colors probably cannot be distinguished until the age of a year and the ability increases very slowly with many children. Yellow, white and red appear

¹ Wien, klin. Wochens., 1905, XVIII, 567.

² Gaz. des malad. infant., 1903, V, 277.

to be recognized before green and blue. In later childhood the power of sight is unusually strong, and light can be endured better than in adult life. Hyperopia seems to be the normal condition in the new born.

Hearing.—This is absent on the day of birth, due probably in part to the filling of the tympanic cavity by mucus and swollen mucous membrane, in part to the approximation of the walls of the meatus. In a few days, however, air begins to enter the cavity and infants can then be awakened by loud noises. In the early months the sense of hearing is very acute and sleeping children are very easily awakened by noise, and they are especially sensitive to high and shrill tones. Infants of 3 months can generally recognize the direction from which sound comes and may turn the head toward it.

Musical tones are sometimes recognized between the ages of 1 and 2 years, and a child of 2 years may distinctly prefer one tune to another and may even know it by name. Very often, however, the ability to recognize tunes does not come until later in childhood, and sometimes never.

In later childhood the hearing is particularly acute, and very weak or very high tones are detected which an adult cannot hear at all.

Smell.—The sense of smell probably exists in the new born, but is certainly slight, although infants born blind are said to be able to recognize the odor of milk. Except for the ability to differentiate pleasant from distinctly unpleasant odors, the sense of smell develops, on the whole, slowly, and is not fully present until later childhood.

Taste.—This is present at birth and the new born can distinguish pleasant and unpleasant substances, such as sweet and bitter. Although the sense of taste during infancy does not always appear very keen, many infants taking without objection medicine which is generally considered decidedly unpleasant, this probably depends, as Preyer¹ has pointed out, on the fact that the taste for sweet is so remarkably developed that the infant often willingly takes anything to which sugar has been added in considerable quantity.

Touch.—This is present at birth, and the touching of the eyelashes, the lips or the hands promptly causes reflex movements. It is, however, much less strongly developed than later. Preyer found the forehead and the external auditory meatus especially sensitive. In older children the tactile sensibility is very acute. The sensibility to *pain* is quite distinct in young infants, but comparatively poorly developed if the area affected is small. Thus the pricking of the finger or toe during a blood-examination, or the scratching of the skin in vaccination frequently produces no crying.

The *temperature sense* in general, though not well developed, is still probably present in the new born as is shown by the comfort a warm bath gives, and the crying produced by chilling of the surface. In the mouth the temperature-sense is active from birth, as evidenced by the refusal of many infants to take cold milk, while warm is readily accepted.

Mental Powers.—The infant at birth is largely in a vegetative state, and its mental powers are dormant. On the whole its sensations are probably pleasurable, or at least not disagreeable, and those which are not are evidenced by a cry. Hunger, pain, cold, lack of sleep and the like are expressed in this manner without the infant being actually conscious that anything ails it. In the 2d month it expresses pleasure

¹ Die Seele des Kindes, 90.

by smiling, as when tickled; but smiles before this age are merely reflex, and often indicative of pain. It is not until the age of 5 or 6 months that the average baby really laughs. In the 3d month there is distinct evidence of mind and thought. At this age the first signs of memory are witnessed, the child clearly recognizing its mother by smiling at her approach, or by ceasing to cry from hunger when preparations for nursing are witnessed. In the 3d or 4th month certain tones awaken its attention and it is interested in bright and especially in moving objects. It also shows its mental activity by grasping after objects, and by attempting, if it reaches them, to put them into its mouth. Before the age of 6 months the infant indicates its recognition of other persons than the mother by smiling at them and realizes the difference between strange and familiar places. When 9 months old it will stretch out both hands intelligently, or will give its hand when told to do so, and enjoys a game of "peep-bo." It clearly understands many things spoken to it, even before it is able to speak any words itself. By the completion of the 1st year it has learned distinctly to indicate by expression of face and by gestures its likes and dislikes for the persons and acts of others.

In the 2d year the baby has some idea of numbers. Sensations of joy, anger, fear and the like are well shown, but none of these make more than a most transient impression, and the child quickly passes from one to the other. In fact, memory in infancy and early childhood is but weak. Later in childhood, however, it is at its highest point.

Speech.—All early sounds made by the child are impulsive. In the 2d month the child often begins to use certain tones of voice, frequently of a "cooing" character, to express comfort, but these are still automatic in nature. About the age of 3 or 4 months the infant commences to utter a few different vowel-sounds preceded by certain consonants, especially m, and b; then d, p, n, and j. These sounds are still not in any way imitative or even selected; but by the age of 8 or 10 months several such syllables are pronounced with some evidence of intent, and by the end of the 1st year "mamma," "papa," and even some other words may be spoken intelligently. At 18 months the infant can express by gestures and a few words many of its desires, and by 2 years it employs very short incomplete sentences of two or three words, using nouns and verbs. Qualifying words of speech are learned later. The time at which speech is first acquired is, however, open to great variation within entirely normal limits.

CHAPTER II

HYGIENE

Prenatal Hygiene.—The health and manner of life of the prospective mother exercise enormous influence upon the well-being of the future child. Constitutional diseases, such as syphilis, affecting her and all acute or chronic maladies require treatment. The general hygiene must be overseen, and especially must the amount and nature of amusements and of exercise taken be carefully supervised. Thus the diet must be generous and digestible; the dress one which does not constrict; the condition of the breasts and nipples carefully attended to before the birth of the child; violent or sudden movements avoided; sufficient outdoor life obtained; the nervous system maintained in a quiet state, and, in general, the hygienic instructions followed which are better detailed in works upon obstetrics.

First Care of the New Born.—In from 5 to 10 minutes after birth, as soon as pulsation has ceased in the cord, a ligature of sterilized surgeon's silk is applied about $1\frac{1}{2}$ inches (3.8 cm.) from the abdomen, and the cord then cut. (For further dressing of the cord, see p. 72.) The child is then wrapped in a soft and warmed blanket and laid in some warm and safe place for a short time until its toilet can be commenced. When ready for this the monthly nurse seats herself on a low chair beside the baby's bath-tub, taking the child, still in its blanket, into her lap, and having the vessels of hot and cold water, the bath-thermometer, and other required articles close at hand. All draughts should be cut off by the use of a screen and by closing doors and windows, and the bathing done before an open fire or other source of heat, unless the weather is very warm. The eyes also should be protected against bright lights. The surface of the body is now rubbed with white petrolatum or olive oil to soften the vernix caseosa, particular attention being given to all the creases and folds in the skin. Next the eyes are washed with a saturated solution of boric acid squeezed into them from absorbent cotton after separating the lids. Should the mother have had a suspicious vaginal discharge, a few drops of a 1 per cent. solution of nitrate of silver should be instilled with a dropper and this washed out later by normal salt solution; and it is safer to use this or to instill a stronger solution of argyrol in every case. The mouth is now washed *very* gently with absorbent cotton wrapped around the nurse's finger and moistened with warm sterilized water, and the nose gently cleansed, as far as possible, with moistened cotton. The face is washed with warm water applied with a sponge or wash-cloth, but without soap, and is then dried with a soft towel. The scalp is next soaped, washed and dried. The toilet of the head being now completed, the rest of the body is rubbed with soap and water, and the baby then placed in the tub filled with water at 100°F. (37.8°C.), kept there for a minute or two, and finally removed to the nurse's lap, where it is wrapped in a fresh flannel blanket, or in the flannel apron which it is advisable she should wear. Here it is patted thoroughly dry with soft towels, particular attention being given to all the folds and creases of the body, these parts being finally powdered slightly with an unscented talcum powder. The child is

then dressed and placed in its bed. Should it seem chilled, as shown by blueness and coldness of the extremities and nose, it should have hot bottles put about it, using great caution against burning it. During the toilet it is important to keep the temperature of the bath uniform by adding hot water as required.

In the case of premature or weakly children it is best to omit bathing entirely until the vitality has become greater, and to substitute rubbing every two or three days with warm oil or petrolatum. (See p. 256.)

Certain matters appertaining to the child's toilet must be considered more in detail:

Bathing.—Succeeding baths resemble the first, except that the oiling is omitted. In place of the tub-bath, however, only a daily sponging is given until the cord has separated in order that the dryness of its dressing shall not be disturbed. Throughout infancy and childhood the bath is given daily, either as soon as the child awakens in the morning or before the morning nap, but never soon after eating. The duration of immersion varies from 1 or 2 to 5 minutes, enough water being used to cover to the neck when the baby is in a semi-reclining position. The nurse, sitting on the right side of the infant, holds it in the tub by grasping its left shoulder and arm with her left hand, thus supporting its head and back on her left forearm. In some cases the reaction after the bath is unsatisfactory. It is then better to employ sponging only.

The temperature of 100°F. (37.8°C.) of the first full bath may be diminished gradually, until, when the age of 6 months or a year has been attained, it is from 90° to 95°F. (32.2° to 35°C.) in winter, or 85° to 90°F. (29.4° to 32.2°C.) in hot summer weather, the reaction of the child always being the guide. In the 2d year the temperature may be from 85° to 90°F. (29.4° to 32.2°C.), according to the effect on the child. After the 4th year the morning bath may be from 75° to 80°F. (23.9° to 26.7°C.) given as a sponge, shower, or tub-bath, with the duration brief, the room warm, and the drying vigorous. In this way it is generally a useful tonic. The temperature of the water should always be determined by a bath-thermometer; not guessed at, as is too often the case.

The *bath-tub* is commonly of painted metal, oval in shape. For the sake of greater convenience to the nurse or mother it may be placed upon a low stand when the bath is given, or supported upon cleated slats laid across the stationary bath-tub of the house. A very convenient tub is a folding one of rubber.

As both hot and cold water, or fresh supplies of water, are often needed a *double sponge basin* with a partition separating the two portions is a very useful article for the baby's toilet.

The *baby's basket* is employed to contain many of the articles commonly used in the toilet. It holds soap, hair-brushes, sponges, powder, and the like, and a certain amount of clothing. A *wash-cloth* is best suited for applying soap. It should be very soft, of flannel, diaper cloth, or cotton stockinet. All folds and hollows of the surface should be thoroughly washed, but no effort made to cleanse the auditory canal.

For the removal of the soap a *sponge* is to be preferred, as water can be more easily squeezed from it upon the body. It should be of fine texture and free from all silicious particles. The sponge and wash-cloth may be kept in the pockets of the baby's basket, but only after they have been thoroughly dried.

The *soap* employed should be unirritating, and free from excess of alkali. Some of the unmedicated superfatted soaps are serviceable.

Imported castile soap is an old favorite. All soap must be used cautiously, lest the skin become irritated.

Towels should be soft and absorbent. Well-washed and, preferably, old diaper-cloth constitutes one of the best materials. Later in life Turkish towelling is excellent. Young infants should be patted dry and then rubbed with the palm of the hand. Later, more vigorous drying with the towel may be employed. As the skin of the infant is extremely sensitive, dusting it with some absorbent *powder* after bathing is advisable, especially in all the folds and hollows. For this purpose talcum or starch is useful. The addition of any perfume is unnecessary and not advisable. Occasionally the application of a very small amount of petrolatum is advantageous if the skin seems too dry.

Local Toilets.—The *eyes* of the young infant should be washed daily with boric acid solution during the first days of life. They should be carefully protected against excess of light on account of the sensitiveness toward it which exists especially in the new born, even into the 2d month of life and longer. Later in infancy and childhood care must still be taken that the child does not injure its eyes by light too intense, insufficient, or badly placed, and the possibility of errors of refraction existing must be borne in mind, lest serious trouble arise. The *mouth* of the young infant may be very gently washed once or twice a day, with absorbent cotton wrapped around the little finger and moistened with sterilized water, but this should be done with the greatest caution, inasmuch as the mucous membrane of the mouth in infancy is extremely sensitive. Many physicians are opposed to cleansing the mouth at all, unless evidence of disease develop.

After the first teeth appear, the mouth should be washed and the teeth themselves rubbed with a moistened cloth morning and evening. When most of the temporary set are cut, a small tooth-brush of softened bristles is to be preferred to the cloth. Occasionally the use of a carbonate of lime tooth powder on the brush, or even of powdered pumice-stone applied with a match stick, is required if stains appear on the teeth. As early as possible older children should be taught to use the tooth-brush, and to draw floss silk between the teeth after each meal. All decay, even of the first set, must be watched for, and the services of a dentist obtained at once, since not only are the carious teeth unsightly, but they cause toothache, occasion indigestion, and even interfere with the eruption of the second set in the proper position. It is especially to be remembered that the permanent anterior molars may appear and even decay, the mother meantime mistaking them for the teeth of the primary set. (See Dentition, Vol. I, p. 37.)

The *scalp* should be soaped daily for some months at the time of the general bath. After the age of 6 months, however, it is not desirable to use soap so frequently, lest the hair be made dry and brittle. In childhood soap must be used occasionally, and water daily, even with the longer hair of girls. The first brush employed should be of camel's hair. Later, when the hair grows coarser, a stiffer brush is required, in order to remove all scalliness. Combs should always be used with great care, and only for parting the hair. In infancy they are not needed. Hair should be trimmed frequently, and even in the case of girls be kept short until well into early childhood.

The *nails* of the fingers should be cut often even in young infants, and be kept clean with a soft nail-brush. The toe-nails should never have the corners rounded off, lest ingrowing follow.

In addition to the daily general bath, the region of the *anus* and *genitals* should be washed with water, without soap, after every movement of the bowels during infancy. If there is much irritation of the skin, starch water may be substituted with advantage. Daily, too, the prepuce ought to be fully retracted and the glans washed carefully with soap and water. Adhesions between the prepuce and glans are very common. These are usually readily broken by "stripping," if this procedure is done early. (See Adherent Prepuce, Vol. II, p. 212.) The labia majora should be separated at the morning-bath and the vulva washed carefully, the direction of the washing being *toward* the anus in order to avoid as far as possible the forcing of feces into the vagina.

CLOTHING ¹

The great requisites for the clothing of infancy are softness, lightness, warmth, looseness, and simplicity. With the exception of the diapers, woollen or partly woollen stuff is the best for use next the skin, except in hot summer weather, or for infants whose skin is especially sensitive, when cotton, silk or linen is to be preferred. The weight of the garments must, of course, vary with the season of the year, but it is important even in winter not to have these of such a weight that perspiration is readily produced. Extra warmth is easily obtained by the use of sacks and the like, which can readily be slipped on or off as required. More children are dressed too warmly than the reverse. The underclothing should cover the whole body except the head and the hands in order to prevent sudden chilling after perspiration. There should be no pressure which can be avoided. All petticoats should be suspended from the shoulders, and should be simple in construction, fastening with but few buttons or with a narrow ribbon. The old-fashioned "pinning blanket" is to be condemned.

First Clothing for Infancy.—Considering the garments more in detail, those required for infancy are as follows:

1. An *abdominal band*, which is of flannel, and is wrapped about the abdomen next to the skin. It holds the dressing of the cord in place. After the falling of the cord the wearing of any band by healthy infants may be omitted entirely, but it is generally advised to replace the flannel band by a knitted circular one of wool or of wool and silk, which is pinned to the diaper and supported by shoulder straps.

2. A *diaper*, which should be of cotton or linen diaper-cloth or still better, of cotton stockinet. A small diaper square may be placed within the outside diaper in such a position that it will receive the urine and feces. This greatly lessens the thickness and consequent heating. A rubber or other impervious cover should never be employed. The diapers should be changed as soon as wet by urine, and should never be used again until after they have been washed. If this precaution is not observed chafing is likely to result. No soda should be employed in washing them.

3. High crocheted or knitted *socks* of silk or woollen yarn.

4. A loose *shirt*, long sleeved and extending below to the band, with the opening the whole length of the front and fastened by small flat buttons, or overlapping well in front and secured by tape. This should be made of all wool, wool and cotton, or wool and silk, and be of thick-

¹ Fuller details concerning the clothing, the layette, etc., may be found in the Author's "The Care of the Baby," published by W. B. Saunders Co., Phila.

ness varying with the season. In the hottest summer weather it may need to be entirely of cotton.

5. A sleeveless *petticoat* of white flannel throughout, made in one piece from neck to hem, fastened at the back with one or two flat buttons, and extending not more than from 6 to 10 inches (15 to 25 cm.) below the feet. For summer the petticoat may consist of a flannel skirt attached to a loose cambric waist. A second petticoat of cambric is often worn over the first to prevent the flannel showing through, but this is unnecessary. In very warm weather the flannel petticoat may be discarded.

6. A *slip* or *dress* of nainsook or lawn, loose, with long sleeves, and opening at the back like the petticoat and of the same length, or only a trifle longer.

A once popular costume known by various proprietary names is similar to that described except that the knitted, close-fitting shirt is replaced by a long loose garment very similar to, and almost as long as, the petticoat described, but with long sleeves. This shirt, the petticoat and the slip may be fitted together, one within the other, and then all three slipped on at once, thus saving considerable turning of the baby back and forth while being dressed. This style is only suitable for long clothing, as the short clothing of later infancy allows too much air to enter under the loose inner shirt.

Besides the garments described the infant needs a *shawl* or a *shoulder blanket* of flannel, to be used as a protection when taken out of the room. Sometimes a thin, knitted worsted *sack* is useful if the room happens to be cooler than usual. This allows for movement of the arms. A *flannelette wrapper* is also often serviceable to put on before the child has its bath. For use out-of-doors there is required for winter a warm long *cloak*, warm *hood*, and a *veil*. In hot summer weather only a cambric or thin silk cap is required.

At night the child should be dressed in a fresh diaper, band and shirt, and then have put on a long, roomy *nightgown* of flannel, cotton flannel, or stockinet, closing with a drawing string at the bottom. In summer it may be of muslin, and need not be fastened below. No socks are required at night.

Dressing the Cord.—Before the clothes are put on for the first time the stump of the umbilical cord must be dressed. It is dried as far as possible, powdered with bismuth, boric acid, or a mixture of salicylic acid and starch and wrapped thickly in salicylated absorbent cotton. It is then laid against the abdomen, a thin compress put over the navel, and the binder applied. The wrappings of the cord should not be changed until the cord falls, provided there is no evidence of putrefaction. The greatest care should be taken to prevent the soiling or wetting of the dressing. The ulcer remaining after the separation of the cord should be dressed with powdered boric acid applied on a pad of absorbent cotton.

Method of Dressing.—After the morning bathing and drying, the nurse, still holding the infant lying on its back in her lap, puts the band about it, or, if this is a knitted one, slips it on over the feet. The diaper is next placed under the buttocks and the ends brought around in front and fastened with a large safety pin to each other and to the tab of the band. Neither band nor diaper must ever be so tight that the hand cannot readily be slipped between it and the skin. The socks are then drawn on. The petticoat is next adjusted inside of the dress, and the two slipped on together over the feet. The baby is now laid upon its

abdomen and these garments buttoned. After the child is old enough to sit alone they may conveniently be slipped on over the head.

Short Clothes.—At about the age of 6 months, the choice of the time depending upon the season of the year, the infant is put into *short clothes*. Its costume then consists of a band, diaper, shirt, petticoats and slip similar to those described, except that the skirts reach only nearly to the ankle, and that it is very customary now to make the flannel petticoat always with a muslin waist and to have a second white petticoat over this. In addition, the child requires *stockings* and *shoes*. The stockings should be white, in order to avoid any action of irritating dyes, and made of silk, woollen or partly woollen goods, or, in hot weather, of cotton. They should always be long enough to reach to the diaper, to which they may be pinned, and should be loose and with broad toes, in order to prevent undue constriction of the feet.



FIG. 13.—IMPRINT OF FOOT.
From life, three-quarters natural size.

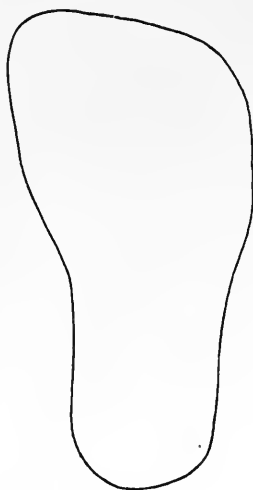


FIG. 14.—OUTLINE OF SOLE OF
SHOE TO COVER FIG. 13.

As the child becomes more active there is often great difficulty in keeping the diaper from falling off. Careful pinning to the band will obviate this, or the infant may wear *diaper suspenders*, or a small light waist to which both diaper and stockings can be attached, the latter by means of elastics or tapes.

The first foot-covering may be moccasins of kid, chamois leather or felt. When the child begins to stand and creep true shoes are required. These may be of soft kid, with kid or thin leather soles. From the beginning they should be rights and lefts, conformed to the natural shape of the foot and with broad toes and without heels (Figs. 13 and 14).

The clothes for the night are the same as in earlier infancy. For use out-of-doors the coat must be shorter after the child has learned to walk, and warm knitted or Jersey *leggings* are serviceable in winter.

In addition to the articles mentioned the infant now needs quilted *bibs* to catch the overflowing saliva. Later, a serviceable article is a *creeping apron*. This should be large and roomy, opening at the back but closed at the bottom, except for the apertures for the legs which fasten just below the knees. Rompers may be used in place of the apron.

Creeping aprons which go over the clothes and fasten under them around the waist are to be avoided, as they allow too free access of air to the legs.

Clothing of Childhood.—At 2 years of age, or less, when the diaper can be dispensed with, the clothes of *childhood* replace those of infancy. The binder is abandoned, if it has been worn hitherto, and the clothing consists of the following articles:

1. An *undershirt*, long-sleeved and high-necked, of material as before and of thickness varying with the time of year, but never too heavy.

2. *Drawers*, close-fitting merino for winter, looser and of muslin for hot summer weather.

3. *Stockings*, long at all seasons. Nothing is gained and much damage may be done by dressing little children in short stockings and leaving the legs bare.

4. A white *muslin skirt* without a waist.

5. A loose, high-necked, sleeveless *waist* provided with buttons, to which the stocking supporters, drawers, and skirt can be attached. If desired, the white skirt can be made attached to a waist of its own, but this offers no advantage.

6. A *flannel skirt* with muslin waist, to be worn in winter.

7. A *dress*, which commonly indicates by its style the sex of the wearer.

8. *Shoes*, which should have the posterior portion of the sole made slightly thicker—i.e., with a “spring”—when the child is 3 or 4 years old, but which should have no true heel until the age of 10 or 11 years.

The clothes for the night consist of a *shirt* and *night drawers*, the latter having closed feet if the child sleeps restlessly and displaces the bed-clothes. The material may be cotton-flannel or stockinet for winter, and muslin for summer. Only in later childhood does the girl begin to use a nightgown and the boy a night shirt; or pajamas may be worn by either sex. When out of doors the hood should be worn in winter until the age of 2 years at least.

At the age of about 2 years or earlier some distinction of sex is made in the style of the clothing, and by 3 or 4 years the boy assumes the ordinary boy's clothes. The girl should not wear corsets or corset-waists as long as she can be prevented from doing so.

SLEEP

Hours for Sleeping.—A healthy infant, in the first few weeks of life, sleeps nearly all the time; in all from 19 to 21 hours, rousing only when being nursed, washed and dressed, or when it is hungry or uncomfortable. As it grows, it requires less and less sleep, and at the age of 2 months, will often lie awake quietly for an hour or so at a time, and show a tendency to some regularity in the hours of sleeping. By the age of 6 months, 16 to 18 hours are required daily; by 1 year, 14 to 16 hours; at 2 to 3 years, 12 or 13 hours; at 4 to 5 years, 10 or 11 hours; at 12 to 13 years, 9 or 10 hours. Sleep during infancy is always easily disturbed by light, noise, and handling. In children it is deeper. From the beginning, the child should be taught to sleep at regular times, and to be put to sleep in proper ways. Walking the floor, rocking, singing to sleep, and the like, are entirely unnecessary. They establish the child in a bad habit, and make a slave of the mother. If the infant is certainly well, it should be put in its bed at the time for sleep and left alone in the room. If it wakes in the night it must not be taken from its bed unless

it is time to nurse it, or the diaper requires changing. Knowing no other method it will soon content itself with this. Allowing a child to go to sleep while nursing at the breast or bottle and before it has finished should be prevented as far as possible. Before the age of 3 or 4 months the baby is made ready for bed at 5.30 to 6 p.m., and should rouse but once or twice during the night. During the day it may sleep at first all it will, but must be wakened for feeding when this is due. This is a matter of importance, as otherwise there can be no regularity in the feeding hours. After this age it may be put to bed at from 6 to 7 p.m., be wakened at 9 or 10 for feeding, as long as this is required, and be trained, as soon as possible, to sleep without further rousing until 6 or 7 a.m. It will now be awake for longer periods in the day time, and by the age of 6 months or earlier will content itself with a nap in the morning of from $1\frac{1}{2}$ to 2 hours or more, and a shorter one in the afternoon. The nap in the day should be of regular length, and the child not allowed to sleep over the time for feeding. At the age of from 1 to 2 years the afternoon nap may be omitted, unless the child seems to require it; the morning nap lasting usually 2 hours. After 2 years the morning nap may be shortened to $\frac{1}{2}$ or 1 hour. The child should continue to take it up to the age of 4 years, if possible. Children of 4 years should go to bed at 8 p.m. or earlier, and the time be gradually changed to 9 p.m. by the age of 10 to 12 years.

When the infant begins to take a regular morning nap, it is best that it should be undressed for it, and be put to bed as though it were night. Often the morning dressing may conveniently be delayed until after the nap, and the morning bath then given. The hour for the morning nap will depend partly on the disposition of the child, and partly on the season of the year. A portion of the day should be selected which will not interfere with the daily outing. A certain degree of latitude is therefore to be allowed in the fixing of the time and in the necessary duration of sleep. The two great desiderata sought for are, first, regularity, and, second, the obtaining of the long quiet sleep at night.

When asleep, the child may assume any position most comfortable to it. In the case of young infants the necessity of the position being changed from time to time must not be forgotten.

Place to Sleep.—The infant should not sleep in the bed with its mother. There is danger of her overlying it, to say nothing of the constant temptation to nurse the child too often. It is liable, too, to receive too little fresh air, as a result of getting its head under the bed-clothes. The first bed generally used is the *bassinette*. It consists of a wicker-basket with high sides, lined, best unprovided with curtains, and with or without a hood at one end. It should be high enough above the floor to escape the draughts. A *cradle* may be used instead, but should be one not capable of being rocked. The *bassinette* is preferable to the *crib* for the early months of life, as it gives the child more support and keeps it warmer. At the age of 8 or 9 months and up to that of 5 years a *crib* is used. This should have sides, which let down on hinges or slides and are high enough to prevent the child falling out, and be provided with a woven-wire mattress. A very serviceable device, especially for sleeping out-of-doors in summer, is the baby *cariole* (Fig. 15).

The *bedding* for the bassinet and crib is the same. There should be, namely, a thin, hair-mattress, a rubber cloth, in the case of infants, to go over this, and a doubled sheet. Sometimes a quilted bed-cover may be put under the sheet to increase the warmth in winter time. The

pillow should be small; a soft, thin, feather pillow with a linen pillow slip. A curled-hair pillow may be substituted in summer if desired.

The *coverings* of the child when in bed should consist of a muslin sheet, as many light blankets as needed, and a light spread. A down quilt is an advantage in winter. Owing to the restless sleep of so many children, some form of bed-clothes-fastener is desirable. A great many children are covered far too warmly at night, with the result that sleep is rendered restless, free perspiration occurs and cold is very easily taken.

It is theoretically better that the infant from the beginning sleep in a separate room from its mother, under the care of a competent nurse. Often this cannot be arranged for many reasons. After the age of a year, it certainly should have, if possible, a separate room at night. Older children are preferably placed in individual beds.

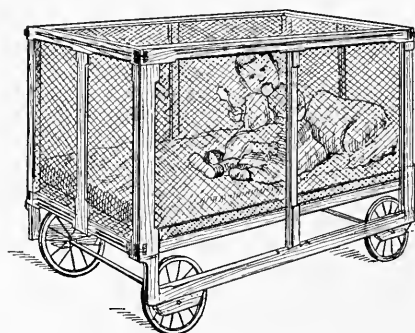


FIG. 15.—BABY CARIOLE.

AIRING, EXERCISE, AMUSEMENTS, AND TRAINING

Airing.—With regard to airing, no absolute rule can be formulated. At 2 weeks of age the nurse may take the infant into another room of a somewhat cooler temperature, proper protection being given by dressing it in its out-door clothing. By the age of 1 month it may be taken into the open air for 10 or 20 minutes, and longer on subsequent visits, until finally it is out of doors 2 or 3 hours or more daily. This applies to the spring and autumn, but in warm summer weather it may go out even at an earlier age, while in midwinter it cannot do so until it is 3 or 4 months old or possibly even later. In place of this it may have its daily airing in a room, the windows of which have been open for a short time, but closed just before the child enters it. Later, when accustomed gradually to the outer air, the windows may be left open during its presence. When possible, autumn babies should be gotten out of doors before the winter sets in. If this cannot be managed, the use of the room with open windows may often be advantageously continued until spring. The important matter is that the infant shall be accustomed *gradually* to the cool air, and that as soon as possible it shall have an abundance of fresh out-door air.

It is a mistake to take an infant into the open air every day no matter what the character of the weather. Very damp, windy, or intensely cold days are to be avoided and the outing in the room with open windows employed instead. Always the condition of the hands and feet and the color of the face are to be watched, and the airing stopped if the infant shows the least chilliness.

Although the bed is usually a better place, there is no reason why a child may not sleep out of doors in warm weather, or even in cold if it is well protected and its condition carefully watched. Some children invariably fall asleep when taken out. Certainly in any case the sleeping-room should have fresh air in abundance.

The first going out should be in the nurse's arms, as this gives greater protection and warmth. After the age of 3 or 4 months, depending upon the season, a perambulator should be used. This should have a sunshade or hood which is lined with some dark color, such as green or brown, to avoid injury to the eyes by the reflected or transmitted light. The infant should recline in the coach, but after the age of 7 or 8 months may sit supported a portion of the time, care being taken that the back does not become fatigued.

Exercise.—After the first 2 weeks of life the infant begins to move its arms and legs freely. It can be assisted in this by having the clothing loose and not too long. At 2 weeks it should be exercised by being systematically carried about in the arms several times a day, lying on a pillow in order to give support to the spine. At the age of 1 month the pillow may be discarded. At 3 or 4 months the infant should be carried about, seated upright in the nurse's arm with her hand supporting its back. It should never be held always on one arm, or lateral curvature of the spine is liable to develop. The carrying about by the nurse is a matter of vital importance. Infants who lie too much in their cribs do not thrive as they should. At about 6 months of age it may be propped with pillows in a sitting position, but only for a little while at a time. From the age of 3 or 4 months on the child may be placed at times upon a thick blanket or mattress, spread upon the floor or elsewhere, securely out of draughts. Here it can make all movements of which it is capable, and has also a good chance later to creep as soon as it is able. The great likelihood of the existence of draughts upon the floor, especially in cold weather, must never be forgotten. Children who have learned to creep or walk will take all the exercise they need. The baby should take its own time to learn to walk, and no appliances to aid it in this are advisable. The legs should be carefully watched for evidence of bowing. (For the age for creeping and walking, see p. 65.) The perambulator or some form of baby-coach must still be used for most of the airing until the age of 2½ or even 3 years, the child being allowed to walk when it desires, but for not too long at a time, lest fatigue ensue or the arch of the foot be overstrained.

Various different forms of exercise will be advisable as the child grows older, in order to bring into play different muscles of the body and to preserve symmetry in development. Little restraint need be imposed unless the child is delicate, or of the nervous and excitable nature which disposes it to exercise to the point of exhaustion. All out-door sports should be encouraged for both sexes. The tricycle and similar apparatus can be used early, and later the bicycle. Swimming, skating, riding, tennis, and the like are all excellent. Jumping rope is harmless unless much overdone. In winter, especially in cities, it is often difficult to obtain sufficient exercise. Dancing and work in a gymnasium now become the best substitutes.

Training and Amusements.—Teaching the control of the bowels and bladder should be commenced early, certainly by the age of 3 months. If the infant is systematically supported on a receptacle at the time when the evacuation of the bowels is found most likely to occur, generally after

a meal, much may be accomplished. The control of the bladder is more difficult to acquire, but even before the end of the 1st year some infants can be taught it, at least during the waking hours. Patience and perseverance are required, and punishment at any age is out of the question.

Amusements begin early, and those may gradually be chosen which educate the mind to a certain extent. Yet the training of the mind must always hold a secondary place, lest overstimulation of it result. In fact all amusements which cause much excitement are to be avoided, and this is especially true if they have been permitted just before bedtime. Insomnia is a natural result. By the age of 5 or 6 months a rattle or a rubber doll will be enjoyed. Later more toys are needed for the house, and others to be used out of doors. Mental and moral training can soon be commenced by using judgment in the selection of the amusements. Thus the constant providing of new toys teaches a child discontent, lack of valuation and lack of care of those which it possesses as well as lack of neatness. Pernicious, too, is the constant effort of the mother to amuse the child, since it should be taught to amuse itself. Many toys may be chosen which instruct, such as picture books, Noah's arks, picture blocks, and, later, lettered blocks.

The inculcating of obedience, unselfishness, absence of self-consciousness, fearlessness, and general kindness to all created things can hardly be commenced too early. There is no hurry about teaching a child to talk, but the use of "baby-talk" in addressing it is always to be avoided.

Girls and boys should play together and at the same games as long as disposed. They have a natural disposition to do this until the age of 9 or 10 years is reached unless they have been taught to do differently. Yet a careful, unobserved supervision should early be kept over the play, since sexual innocence in early years is by no means so common as often assumed.

School Life.—Most important is it that the mental powers shall not be forced. Precocity is not desirable. The kindergarten is excellent for young children, and much instruction is gained in this way. Yet even this is sometimes too stimulating. There is nothing gained in having a child learn to read before the age of 6 years. Even at this period the hours spent in school are generally far too long. From 3 to 4 hours daily from the age of 6 or 7 up to that of 10 years are quite enough. Ocular defects, lateral curvature of the spine, loss of appetite, poor general health and nervous irritability are common results of improper school life.

As to the fittings of the school-room, the chairs employed should be low enough to allow the feet to rest comfortably on the floor, and should be so constructed that they support the *lower* part of the back. The desks must not be high, or the child's eyes are brought too close to its work, and the spine is liable to be distorted when it writes. The light must be so disposed that there is no glare in front, and that trying cross lights are done away with. There should be at least 300 cubic feet (8.49 cubic metres) of air space for each individual, and, in addition, the air should be constantly and completely changed by ventilation several times an hour.

NURSERY

Day Nursery.—Inasmuch as the baby spends so much of its time in the nursery, the *position* of this in the house is of the greatest importance. It should be selected with reference to winter rather than summer, since

the child will be out of doors so much of the time in the latter season. It ought to be, if possible, the brightest, airiest room in the house. The preferable exposure is south, or, if this is not obtainable, east rather than west on account of the morning sun. A corner room with south and west windows is ideal. The windows should not extend to the floor, since the children readily take cold as a result of getting too close to them. Other things being equal, the nursery should be on the third floor, if this is not directly under the roof, as it is more out of the way here. It should have at least 500 cubic feet (14.16 cubic metres), and preferably 1000 cubic feet (28.32 cubic metres) of *air space* for every person occupying it, and in addition should be properly ventilated by a constant and abundant supply of fresh air. Window ventilation of some form accomplishes this fairly well yet with some danger of draughts. Probably the best of all ventilation in houses not especially built to secure it in other ways, is obtained by an open fireplace with a fire burning. Sometimes it is safer to ventilate from an adjoining room. In addition to the constant ventilation the room should be aired thoroughly with wide-open windows once or twice a day at times when it is unoccupied.

Draughts upon the floor nearly always exist in cold weather even when the windows are closed, the air being chilled by the cold panes. Double sashes will prevent this to a great degree, but in very cold weather it is better to keep the child away from the windows and even off of the floor.

In the line of *heating* probably nothing equals a hot-air furnace, especially if the air comes from over hot-water pipes, as it is easily controlled and supplies fresh warmed air from without. A fireplace, or a properly constructed coal stove, is effective in the same way, but gives an uneven heat and is less controllable. Gas-stoves or oil-stoves should never be used unless provided with smoke-pipes connected with the chimney. The only exception is for the rapid heating of the room before the bath, when this can be accomplished in no other way. Steam and hot water radiators, though serviceable heating apparatus, afford no ventilation whatever.

All hot registers, fireplaces and the like, and all lights, should be so guarded that a child cannot possibly burn itself. A couple of thermometers should be placed in different parts of the nursery, one of them near the floor, since it is in here that so much of the time is passed. The temperature of the nursery should be as uniform as possible at from 66° to 70°F. (18.9° to 21.1°C.).

The *furnishings* of the nursery may be attractive, yet simple and arranged with a view to cleanliness. The floor should be well made, the cracks stopped, and the whole painted or varnished, but not with a slippery finish. Carpeting in the form of one or several rugs is required. These should not be tacked down. The walls and ceiling are best painted. If paper is used one must be sure it contains no poisonous coloring matter. Windows should have no heavy curtains, and should be fitted with bars to avoid danger of falling out. The doorway should have a swinging or sliding gate to keep the child from the stairway. The furniture of the nursery is better if not upholstered. Chairs with projecting rockers and furniture with sharp corners are to be avoided. Among the articles of furniture is the "nursery chair" on which the infant is placed when it is to empty its bladder or bowels, and only then. Portable screens are very useful. A nursery refrigerator is a great convenience.

The nursery must be kept scrupulously clean and neat. No wet diapers are to be kept hanging about, and no empty nursing bottles

or empty dishes left in it. A stationary washstand may be in the room, provided that one is sure that the trapping is perfect, and that there is no danger of the entrance of sewer-gas.

Night Nursery.—Although a room set apart especially for the night is not an essential, yet it is a great convenience when there are several small children in the family. The requirements are the same whether this separate room or the day nursery is used, and the infant should sleep here whether at night or during its daily naps unless the sleeping in the daytime is done out of doors. The heating and ventilation are provided as in the day nursery, and the room should be thoroughly aired and then warmed again after each occasion on which it is used. There must always be some arrangement for heating food at night, such as an alcohol-stove or small gas or electric heater. The temperature of the room should be about 65° or 70°F. (18.3° or 21.1°C.) during the early weeks and later from 50° to 65°F. (10° to 18.3°C.). A window should be partly open as a rule, but it is not wise to have it widely open during very cold weather. Light should not be allowed to burn all night. If it is necessary to have this on occasions, a small wax night light is excellent. When it can be obtained, nothing equals electric light, as it consumes none of the oxygen. Small shaded electric lamps can be obtained. If oil lamps are used for lighting before the child is put to bed they must be out of harm's way where they cannot be upset. The beds must be carefully placed out of the way of draughts. The use of folding screens is an aid to this end. If the child is bathed and dressed in the night nursery the temperature of the room should be elevated to 70°F. (21.1°C.) before the toilet begins.

Sick Room.—In the case of ordinary slight ailments no special room is required, one of the nurseries being generally employed; but in more serious illnesses, or where special quiet is demanded, the room selected should be away from sources of disturbance. Numerous bottles of medicine should not be allowed to stand about. Not only is this unsightly, but there is danger of giving the wrong drug. Ventilation is very important, yet sometimes difficult to obtain satisfactorily on account of the danger of draughts in certain diseases. Sometimes it is necessary to ventilate entirely from an adjoining room where the air is kept fresh and warmed.

The special requirements of the sick room for infectious diseases will be described in considering that subject. (See Infectious Diseases, p. 307.)

NURSES

The Nurse-maid.—Two of the greatest requisites in a nurse-maid are intelligence and docility. Consequently the nurse-maid should be preferably in middle life, but better young than old. The old nurse-maids are commonly so opinionated that their methods, generally bad ones, cannot be changed. There are, of course, exceptions to this. The maid should be neat, strong, healthy and of loving and patient disposition. No matter how faithful and efficient she may seem, no mother, even the inexperienced, dare give up her own constant supervision.

The Trained Nurse.—The nurse trained specially for the care of infants and children during illness will be of the greatest service to the patient, the family, and the physician, if with a proper knowledge of nursing she combine a special recognition of the responsibilities and relationships which the nursing of a sick child creates. If she is not so

qualified she may do more harm than good. It is the physician's duty to select carefully the nurse adapted for this service and to supervise the nursing constantly. A few of the qualifications required may be passed in review:

1. The nurse should be accustomed to and have a fondness for little children. Nothing is more exacting and trying than the nursing of sick children, and, unless there exists a special native fitness and liking for it, a nurse should not undertake it.

2. She should be quick to recognize and prompt to report important symptoms. Only careful training and inborn acuteness of observation, combined with good judgment, can give a nurse this invaluable quality.

3. She should be quiet, gentle, firm, resourceful, and comforting. The influence of such a nurse upon the mental state of a nervous infant and nervous mother is often most remarkable.

4. The nurse must carry out the physician's directions implicitly and without criticism before the family; yet know when emergency justifies and compels a failure to do this. Unless she has this quality her actions are too much those of an automaton.

5. She must not be imposed upon by the family. No nurse can do good work who is exhausted by the nursing of a fretful, ill infant, in which she is given no opportunity for systematic rest. Families are prone to forget this, and for the sake of the patient the physician should see that matters are properly arranged.

6. On the other hand, the nurse must be willing and helpful, and avoid making her presence a burden. The installing of a nurse necessarily adds to the responsibilities, expenses, and cares of the manager of the household, and mothers often dread it with reason. If the nurse seems to need some one specially to wait upon her, as is often the case; is not quick to offer to prepare and serve special food for the child; is unwilling to take the ordinary care of the room, or to take the place of the mother in giving a convalescent child its outdoor airing in its coach; is displeased if she is not taken fully into the family social life, as at meals or in the evenings, she fails to recognize the dignity and duties of her profession, and is not a suitable caretaker for a sick child. Inasmuch as these faults are all too common, the physician must acquaint himself with conditions and correct those which are wrong.

CHAPTER III

BREAST-FEEDING

Advantages.—The general superiority of breast-feeding can hardly be questioned. Statistics show that the mortality of breast-fed children in the 1st year is much less than that of those fed on the bottle. (See Mortality, p. 212.) In fact the average probability of death in the 1st year in artificially fed infants as compared with the breast-fed may be placed at 5 : 1. The figures of Camerer¹ and others demonstrate, too, that infants fed artificially do not as a class gain as rapidly in the 1st year as those breast-fed. Further, the interesting studies of Rose² upon 164,000 individuals showed that the deleterious influence of bottle-feeding could often be detected even in adult life. Although artificial feeding carefully carried on will undoubtedly give much better results than statistics indicate, yet the superiority of breast-feeding, as a rule, remains unquestionable. This is a natural and unavoidable result of the fact that other mammalian milks are in a sense foreign substances and cannot be made otherwise. Certainly every effort should be put forth to have the infant breast-fed for the first 3 months of life at least, by which time a fair start has been obtained, and success with artificial feeding is more likely to follow.

Ability of Mothers to Nurse.—The long-continued propaganda for artificial feeding, especially with proprietary foods, had in the course of years the natural sequence that breast-feeding in many localities became comparatively uncommon, or at least continued for a very short time. I have reviewed the subject rather extensively elsewhere,³ and a few illustrative statistics may be quoted from those publications. Conditions vary greatly with the locality. In Japan breast-feeding is the rule. In Greenland artificial feeding is scarcely known, and among the Esquimo of Alaska there is no cow's milk available, and infants are often nursed for from 2 to 3 years. On the other hand, Nordheim⁴ found that only 3.6 per cent. of the women studied in Munich nursed for over 3 months, and only 0.8 per cent. for over 6 months. Neumann⁵ stated that 55.2 per cent. of the children seen by him in Berlin were nursed in 1885 and only 31.4 per cent. in 1890; and Koplik in New York⁶ found only 10 per cent. in private practice fed on the breast alone.

Much of this apparent inability is probably unwillingness and a lack of teaching by the profession, and the indications are that with the increasing urging by physicians upon mothers regarding the importance of breast-feeding the frequency is now again increasing. An investigation by Mitchell⁷ upon 2819 mothers in Philadelphia showed that the average duration of lactation was 6 months. Negris⁸ in Graz found a

¹ Jahrb. f. Kinderh., 1893, XXXVI, 249.

² Deutsch. Monatsschr. f. Zahnkunde, 1905, XXIII, 13. Ref. Ziegenspeck, Verhandl. Gesellsch. f. Geburtsh. u. Gynäk., 1907, XII, 829.

³ New York Med. Jour., 1909, Dec. 4; Journ. Amer. Med. Assoc., 1912, LIX, 1874.

⁴ Arch. f. Kinderh., 1901, XXXI, 89.

⁵ Deut. med. Wochenschr., 1902, XXVIII, 795.

⁶ Journ. Amer. Med. Assoc., 1912, LVIII, 75.

⁷ Jour. Amer. Med. Assoc., 1916, LXVI, 1690.

⁸ Wien. klin. Wochenschr., 1905, XVIII, 459.

physical disability in only 10 per cent. of the women in the obstetrical clinic. Mme. Dluski¹ observed that 99 per cent. of the women seen in the obstetrical clinic of Pinard in Paris could nurse at least for a time; and Blacker² in London found only 2.5 per cent. of the mothers with a physical inability to nurse. That the general ability can increase through the training of the people is indicated by the experience of Jaschke³ who observed that while in 1904, 64.01 per cent. of the infants in Heidelberg were breast fed, in 1907, 86.14 per cent. were so fed, and in some years 97.22 per cent. The frequency of maternal nursing is, however, far from what could be desired; and it is undoubtedly true that the nervously organized mother of the upper classes often is unable to nurse her infant in spite of her strong desire to do so. Yet in numerous cases it is the ill-considered advice of the nurse, or still oftener of the physician, which is the cause of early weaning or of failure to nurse at all. Many a woman whose breast-milk has ceased entirely for a day or two, or in whom it has been slowly diminishing, will still be able to nurse her baby if given proper treatment and encouragement. So, too, the failure of the secretion to appear in the first few days after parturition is no reason for abandoning efforts at maternal nursing, inasmuch as it is not at all infrequent to have the full secretion delayed for a number of days. A scanty supply of milk is no excuse for weaning, since even the small amount, helped out by artificial feeding, is better for the baby than no breast-milk at all. So, too, the apparent fact that the milk disagrees with the infant should be regarded with suspicion, especially in the early weeks after birth, inasmuch as it often happens, when the mother gets out of bed and resumes her ordinary method of life, that both the quantity and the quality of the secretion will change in a satisfactory manner.

Probably one of the chief causes for the early disappearance of the secretion is the failure to obtain a satisfactory emptying of the breast. This is especially liable to occur when the infant is weakly and grows easily fatigued by sucking. Another cause often assigned for failure to nurse is painful fissuring of the nipples. This is a real difficulty, but generally can be overcome with care and patience. Indeed the causes which are assigned for the failure to nurse are often of the most trivial nature. This has been interestingly studied especially by Keller.⁴ In addition in the poorest classes is the very real difficulty that nursing women are often obliged to go to work. This frequently necessitates early weaning. It is a matter which can best be dealt with from a civic point of view, aid being given to mothers in these circumstances.

Preparation of the Prospective Mother.—In view of the importance of breast-feeding the preparation of the prospective mother for this is of great importance. The general hygiene has already been briefly referred to (p. 168). The diet should be rather more abundant than usual, the bowels should be kept regular, gentle exercise in the fresh air should be taken, and simple amusements enjoyed, without much strain of the emotions. Undue pressure of clothing upon any part of the body, and especially upon the breasts, should be avoided during pregnancy. If the nipples are retracted nipple-protectors may be worn. Useful, too, to develop the nipples is gentle traction by the fingers, or, still better in some cases, by the application of the breast-pump several times daily.

¹ Thèse de Paris, 1894. Ref. Marfan, *Rev. mens. des mal. de l'enf.*, 1894, XX, 11.

² *Manchester Med. Chron.*, 1900, Series 3, II, 323.

³ *Medical Klinik*, 1908, IV, 257.

⁴ *Wien. klin. Wochenschr.*, 1909, XXII, 635.

Neither procedure, however, should be practised until the last 4 weeks of pregnancy. At the same time the nipples must be hardened by applying twice a day equal parts of glycerine, tannic acid and water, or a saturated solution of boric acid in equal parts of alcohol and water.

Hygiene of the Nursing Mother.—The care of the nipples during the period of lactation is even more important than before the birth of the child. After nursing they should at once be washed and dried gently and then smeared with a little sweet oil. If they become excoriated or fissured, the application of a small amount of bismuth-ointment (equal parts of bismuth and ol. ricini) or of compound tincture of benzoin is often efficient. Sometimes an initial soreness will pass away entirely in a few weeks. In other cases, where the pain of nursing is intense, the use of an artificial nipple must be tried. Great care must be observed to keep this scrupulously clean.

During the first few days after childbirth the nursing mother should eat rather cautiously, taking small quantities of easily assimilated food frequently, lest overfeeding produce indigestion. There is no need, however, for any great restriction of diet. Toward the end of the 1st week she may have meat and later may, as a rule, eat plentifully of any nutritious, digestible food. In addition to this milk, cocoa, or some other light liquid nourishment should be taken at bed-time and possibly between meals. Weak tea and coffee are permissible. With regard to stimulants, the nursing mother, if in good health, need nothing of the sort. Delicate mothers are sometimes much benefited by one of the malt liquors or extracts, and the quality and strength of the milk increased thereby. Alcohol, however, is to be prescribed with caution lest habits of intemperance be established.

A proper amount of exercise in the open air, taken with regularity, favors greatly the production of a healthy, nourishing milk. Carriage-riding is beneficial, but does not take the place of walking, begun as soon after parturition as the state of the patient permits. Late hours should be avoided, and plenty of rest and sleep obtained. All causes of nervous excitement and of worry should be carefully shunned.

Rules for Nursing.—The infant should be put to the breast after it has been washed and dressed, and as soon as the mother is sufficiently rested to permit it. It lies upon its left side to nurse from the right breast, and *vice versa*, its head being supported by the arm of the mother who rests upon her side or is propped up slightly in bed. When the mother is convalescent and able to sit up, she should lean slightly forward while nursing, partly supporting and steadying the breast with the fingers of one hand, in order to keep its weight from pressing against the infant's nose and interfering with its breathing. If the milk flows too freely it may be restrained by compressing the base of the nipple slightly with the finger and thumb. One breast should be sufficient for one nursing. The giving of both breasts at a nursing is inadvisable, unless a scanty secretion makes this necessary. A good secretion is stimulated by a thorough emptying of the breast; and the giving of both of them is liable to leave neither empty. An infant should nurse for not more than 15 or 20 minutes, and should not be allowed to go to sleep until it has finished its meal. Many infants are uniformly contented with a shorter period, because they have nursed with vigorous rapidity; others stop nursing in a few minutes because of feebleness or because the breast is empty; others from habit or from an insufficient milk-supply

wish to nurse longer. The weighing of the infant before and after nursing will determine the actual condition present.

Intervals.—During the first 2 days but little secretion of any kind is in the breast, the needs of the infant are slight, and it is not necessary to nurse the child oftener than from 4 to 6 times in the 24 hours. The employment of some substitute such as a solution of sugar or a starchy decoction is generally entirely unnecessary, but water should be given freely. If there is no milk by the 3d day feeding with a very weak substitute-milk-mixture must be commenced while efforts at nursing are still continued. Only in exceptional cases, where the infant seems particularly hungry, is earlier feeding required. As has been pointed out elsewhere (p. 21) no real benefit is gained by feeding an infant even with breast-milk of another woman from the 1st day of birth. The infant should be kept very quiet after nursing. A little turning about, jogging on the knee, or other motion may very readily cause vomiting. From the beginning the greatest regularity should be observed in the times for nursing the infant, keeping in mind, too, the fact that at night both mother and child should have as much undisturbed sleep as possible. The duration of the intervals between feedings has been a subject of much discussion. The fact that the healthy, breast-fed infant empties its stomach often within 1 hour after nursing, and practically always in not more than 2 or $2\frac{1}{2}$ hours (see p. 44) is an indication that an interval as long as 4 hours, as recommended by many, may be too great, and that the shorter intervals, which have so long been in vogue, should not be too readily abandoned. The fact is that there can be no absolute rule applying to all infants, although there should be regular intervals for each individual. In the case of a hearty infant taking a large amount of food at each nursing the intervals will necessarily be longer. A more delicate infant with less appetite, or one with a limited gastric capacity, may need more frequent feedings. The following statements can therefore apply only in a general way, being viewed as average figures for average babies: After the first 2 days, in which the child is fed only every 4 or 6 hours, the interval between nursings, *i.e.*, the *beginning* of each nursing, should be from 2 to $2\frac{1}{2}$ hours during the daytime, with only 2 nursings after 9 or 10 p.m. until morning, there being in all from 8 to 10 nursings. This holds good for the first 4 weeks. From 4 weeks to 3 months the interval should be $2\frac{1}{2}$ to 3 hours in the daytime, with only 1 nursing after 10 p.m., the total number being 7 or 8. From 3 months until the age of 4 or 5 months the interval should be 3 to $3\frac{1}{2}$ hours by day, perhaps with 1 nursing after 10 o'clock at night in case the child awakens for it. The total number of nursings is, therefore, 6 or 7 in 24 hours. From this time until the age of 1 year the intervals should be 3 to 4 hours, but no night nursing after 10 p.m. is required, and the total number equals 5 or 6. The following tabular arrangement shows in convenient form the intervals and number of nursings, not including the first two days of life:

TABLE 26.—FREQUENCY OF NURSINGS

Age	Intervals, hours	No. of feedings in 24 hours	No. of feedings between 10 p.m. and 6 a.m.
Birth to 4 weeks.....	2- $2\frac{1}{2}$	8-10	2
4 weeks to 3 months.....	$2\frac{1}{2}$ -3	7-8	1
3 months to 4 or 5 months.....	3- $3\frac{1}{2}$	6-7	1 (2)
4 or 5 months to 12 months.....	3-4	5-6	0

It may be again emphasized that these figures are only a guide, some infants requiring fewer and some more frequent nursings at certain ages. The great principle is, that a rule, once established, and which has been found satisfactory, must be followed with uniformity, unless some very special reason arises for modifying it. The determining of the exact hours of the day for the nursings will depend in part on the time the infant regularly awakens in the morning, and in part upon the hours of its regular daytime naps. The child should be awakened for its food when the appointed time comes during the daytime and at the 9 or 10 P.M. feeding. This will soon train it to waken of its own accord at the proper time. During the night it may sleep as long as it will, the hours for nursing being movable ones.

Amount of Food.—This can be determined with even less fixity than in the case of the intervals, owing to the very different demands of different infants. It varies also from time to time in the same infant. If an unusually large amount is ingested at one nursing, the child will naturally require less at the next one, or perhaps even refuse it altogether. This occurrence need, therefore, be no cause of alarm in the case of a healthy infant. The important consideration is that a sufficient total quantity of breast-milk be taken in the 24 hours. Reference to the table upon page 92 will show what may be regarded as the average amount taken at each nursing and during the whole day. The matter is referred to again in considering Artificial Feeding (p. 133). It is a point of interest, as pointed out by Feer,¹ that in any event the greater part of the milk is ingested by the infant in the first 5 minutes of nursing.

Causes Making Nursing Inadvisable or Impossible.—Many causes may render nursing impossible or harmful. (See p. 103.) Pregnancy is one of the chief of these, the milk becoming insufficient or indigestible, or the secretion disappearing entirely. The return of menstruation is quite frequently regarded by mothers as a necessary cause for weaning. This is a mistake. The milk may be temporarily altered to a slight degree, or may exhibit no change whatever. It is true that such an occurrence sometimes heralds a permanent cessation of secretion, but there is no need by weaning to anticipate this. The development of an acute illness may render a temporary cessation necessary, this depending altogether on the individual case. The effect upon the mother is that to be considered; and a decision against nursing should not be made too hastily. The occurrence of a long-continued illness, such as typhoid fever, generally prohibits nursing, as the milk is liable to be poor and the drain upon the strength of the mother too great. The presence of healed tuberculosis is a contra-indication, chiefly because the drain upon the mother is too severe. Women with active tuberculosis, especially of the lungs, must not nurse, both for their own sakes, and on account of the danger through the close contact of infecting the infant. The likelihood, however, of this infection occurring through the milk is very slight indeed. Sepsis, nephritis, puerperal eclampsia, diabetes, insanity, and frequently repeated epileptic seizures, are contra-indications against nursing. Other chronic illnesses or the existence of a delicate state of health may contra-indicate nursing, both because of the harmful effect upon the mother, and because the milk is liable to be of poor quality. However important breast-feeding may be for the infant, the mother's health should be the first consideration. One must be sure, however, that nursing will be undoubtedly harmful to her. It is true that the strain

¹ Jahrb. f. Kinderh., 1896, XLII, 225.

of nursing often does cause loss of weight and of strength in an anemic or neurotic mother; but it is generally a condition which can be tolerated without any permanent harm, certainly for a time at least, and the effort at nursing ought to be made. Syphilis existing in the mother is no contra-indication to her nursing, since the child is also certainly syphilitic at the time of its birth. So, too, an infant with congenital syphilis may continue to nurse without danger to the mother, since she must be syphilitic also. Mastitis prohibits nursing from the diseased breast as long as the suppuration continues. Retraction of the nipple which cannot be overcome, and fissures which will not heal, causing unbearable pain, often make the continuance of nursing impossible. Sometimes an artificial nipple will overcome the difficulty, and in any case thorough trial of nursing should be made before this is abandoned. The secretion of milk on which the infant persistently does not thrive makes weaning imperative. There exists, however, a far too great readiness on the part of physicians to advise weaning under these circumstances, without sufficiently long-continued efforts to remove the difficulty.

On the part of the infant nursing may be temporarily or permanently interfered with by severe coryza, cleft palate, or conditions of great debility. The last is seen especially in premature infants, or those with congenital asthenia from other causes, the baby being too weak to suck properly. In this event the breast-milk must be fed from a dropper until the child is stronger.

Signs that Breast-feeding is not Satisfactory.—The breast-milk may be insufficient in quantity; the child may for various reasons fail to get enough of what is in the breast; or the milk may be inferior in quality. Not every breast-milk is suitable for the infant even when sufficiently abundant. It is important to determine as promptly as possible whether nursing is satisfactory, as otherwise valuable time may be lost and damage done. The normal infant shows a steady gain in weight according to the rates already given (p. 23). This does not mean, however, that the gain must be regular and unbroken from day to day, for such is by no means the rule; nor does it indicate that a gain which is not quite up to the normal figures necessitates a change in the diet. A baby who is receiving a supply of milk defective in quality or quantity constantly falls behind in its weight-curve and in its appearance of well-being; is liable to become hungry too soon; to cry with dissatisfaction when or before nursing is over; often has too few bowel-movements in the twenty-four hours, and these are frequently constipated. Weighing the baby or the mother before and after each nursing will show whether the absolute daily quantity of milk ingested is insufficient (p. 92). Sometimes a weight-curve which has been entirely satisfactory perhaps for some months makes a decided deviation from the normal during 1 or 2 weeks, yet without there being any other noticeable disturbance in the condition of the child. This is very liable to mean that the maternal secretion is unsatisfactory. In other cases it is evident that the milk produces symptoms of indigestion. Actual vomiting should not occur in the normal infant, unless as a result of its being disturbed in some way. It may not infrequently be on account of indigestion having developed, either from the milk being too abundant or too rich in some respect. For the same reason the stools may be too frequent or not well digested; and the occurrence of colic is a very common evidence of indigestion. If with these symptoms the baby is still thriving normally in other respects, there can be no thought of weaning, although an effort at a modification

of the secretion (p. 106), or of some of the details of the method of feeding should be made, and treatment for the indigestion given. Again, a child may fail to gain on account of the existence of some disease other than a digestive one, and a careful study of the case must be made for some concealed ailment before concluding that the breast-milk is unsatisfactory. The special symptoms produced by an excess of the different elements of the food are referred to more conveniently under Artificial Feeding (p. 127).

MIXED FEEDING

Very many women secrete only enough milk to nourish the child in part. Such circumstances render it necessary either to wean, to employ a wet-nurse, or to supplement with artificial food. The last plan is greatly to be preferred to complete weaning, provided the mother's health is not suffering and her milk is of good quality. To determine the latter the milk should be analyzed and then, if necessary, an attempt be made to improve its quantity or quality. If efforts to improve it fail, substitute feedings should be commenced, at first a little weaker than normal human milk and then, when tolerance of the new food is established, of such percentage-strength as the gain in weight shows to be requisite. So, too, even though the mother's milk may be sufficient, partial substitute feeding may well be commenced if her health is distinctly suffering. The number of substitute feedings, and the time in the twenty-four hours for them, depend always on individual circumstances. They may either replace entirely some of the feedings, the mother nursing at other times from both breasts if necessary, or a small bottle may be given after each nursing. The latter is in many respects the better plan, so far as the baby is concerned, since the more frequent stimulation of the breast by nursing tends to maintain the supply of milk. In fact, the giving of two or more bottles daily without nursing immediately preceding it is extremely liable to hasten an entire disappearance of the maternal secretion. From the side of the mother, however, the giving of one or two bottles daily without nursing has the advantage that it furnishes her with an increased degree of freedom. Systematic weighing, at least twice a week, will be the indication of the success of the mother and of any necessity to wean entirely and quickly. As to the amount to be given at each feeding, this depends upon the age of the child, and the quantity it obtains from the breast. To determine this the baby may be weighed before and after each nursing during one or two days, and then given enough artificial food to make up the total quantity which the age would naturally require in the average infant. (See p. 133.)

It is frequently advised to give a baby one bottle of artificial food from the beginning, in order that it may become accustomed to this in case of the maternal supply failing. This seems like meeting a possible danger by undergoing a real one; and it is not necessary if the infant is early trained to take water from a bottle. The only advantage is the freedom for the mother already mentioned. It is of common occurrence in attempting to employ mixed feeding to find that the infant soon will refuse the breast and take only the bottle, finding nursing from it easier. Judicious starving will generally overcome the difficulty. Sometimes putting a little sugar or some of the bottle-food on the maternal nipple, or the employment of an artificial rubber-nipple, will persuade the infant to nurse.

It does not necessarily follow that mixed feeding, once commenced,

shall continue indefinitely. The mother may be suffering from a temporary impairment of health, or the milk may have diminished as a result of maternal anxiety or other psychic cause; or the difficulty may exist only in the first few weeks of the infant's life. It will then be found, with the increasing supply of milk, that the infant has no desire for the bottle and is yet gaining weight normally, and a purely maternal nursing is thus reinstated. During the period of mixed feeding, however, it is very important to see that the mother's breasts are frequently emptied as thoroughly as possible in order to stimulate the secretion.

WEANING

With very many women the supply of milk is liable to diminish greatly by the time the infant is 8 or 9 months old, or even, as previously stated, before this. Weaning is thus rendered necessary early. When, however, nothing of this sort occurs, the child should be fed on the breast alone during the first 10 or 12 months of life. We should certainly attempt to have maternal nursing continue until the infant is 5 or 6 months of age, after which period substitute feeding will be better tolerated. Nursing longer than 12 months generally offers no possible benefit and is often deleterious to the infant. It is not infrequently practised by mothers in the hope of preventing conception.

Weaning should not be done in hot weather if it can be avoided. At this season, if the child is clearly thriving on the breast-milk and the mother is not suffering, it is well to prolong breast-feeding beyond the usual time for weaning and until the hot weather is over. Exceptionally the reverse is true, and weaning may well be hastened before hot weather sets in. This is, however, not often required, and the step must be taken cautiously. With proper guarding against the employment of an impure milk, a baby may readily be weaned in the summer-time if necessity arises.

Some of the causes which make weaning necessary have already been discussed (p. 86). The sudden cessation of the maternal supply, the secretion of milk which is harmful to the child, or decided illness on the part of the mother, may compel a sudden complete weaning. So, too, the absolute refusal of the child to take anything but the breast may require it. This last is a very real and not infrequent factor in cases where the mother's milk is not entirely sufficient. It is sometimes necessary to withdraw the breast absolutely and almost to starve the child for several days before it will take its artificial nourishment. This can often be avoided by teaching the breast-fed child, early in life, to suck water from a bottle. When, however, such an emergency arises, the bottle should be offered to the child in the mother's absence, as the infant is more prone to be content with it under these circumstances.

Whenever possible, however, weaning should be done gradually. The child should be started at the age of 10 or 11 months with the mixed feeding described. At first only one bottle is given daily, using a formula much weaker than the needs actually demand, and gradually increasing the strength of the milk until one sufficiently nourishing is found to be well tolerated. In a few days another breast-feeding should be omitted and two feedings of milk-mixture given. This process is continued until the infant is entirely on artificial food. After this the strength and quality of the food is to be increased gradually in the manner to be considered later (p. 132).

FEEDING BY A WET-NURSE

Advantages.—The milk of a wet-nurse is beyond question generally the best substitute for mother's milk. In fact in many cases it may be better than the milk of the mother. Many infants have been saved by wet-nursing who would unquestionably have perished if artificial feeding had been continued. This is particularly true of marantic infants with serious digestive disturbances. So, too, the rearing of young infants, normal at the start, with artificial food in the heat of summer, and especially in the cities, is often of such difficulty that wet-nursing is greatly to be desired, and this should certainly be urged by the physician, if a brief trial with artificial food shows any deterioration of health.

Yet, on the other hand, it is a mistake to regard wet-nursing as an unqualified good. Very many wet-nurses are prone to take advantage of their important position, become overbearing and insolent, and be a trial in the household which words cannot describe. The nurse often turns out unreliable and may abandon her position at some most inopportune time. It does not follow, too, that the milk of the wet-nurse will agree with her foster-child. Sometimes milk, at first abundant and good, becomes altered by the changed methods of living which the nurse experiences in her new position or by anxiety or nervousness, although this may be only a temporary matter. For all these reasons mothers dislike the introduction of a wet-nurse into the household. Very many of them, too, have so strong a prejudice against having their babies nursed by another woman, that they prefer seeing them die rather than consent to it, and I have heard them frankly admit this. Others draw the line at the engaging of a woman other than white. All this is naturally wrong and distinctly selfish, since the baby's good should be the first consideration. Equally wrong, and often fatal in its results, is the deferring of the employment of a wet-nurse until it becomes a last resort.

It is sometimes urged that the employment of a wet-nurse is inhuman and selfish on the part of the employer, because it forces the nurse to deprive her own infant for the sake of the foster-child. This is altruistic in theory, but illogical in fact. The wet-nurse's own child is probably in a healthy and flourishing condition, or the woman would not have been selected as a nurse. The foster-baby is probably having dangerous digestive trouble existing or threatening, or a wet-nurse would not have been sought. By employing a wet-nurse there is much greater chance of saving two lives instead of one. — Still more important is the fact that the wet-nurse takes this position to obtain the livelihood of herself and child. If she did not do this, she would be obliged to take up some other and less well-paid work, and in this event she could not nurse her baby.

The Wet-nurse's Baby.—There is no objection to the wet-nurse taking her own baby with her into her new position. In fact, she is less liable to be anxious about it, and this conduces to the maintenance of her supply of milk. On the other hand, it is bad policy, in my experience, to make any arrangement whereby she shall try to supply milk for both babies. The foster-baby is the one likely to suffer in this event. Indeed, the only vital objection against the presence of the nurse's baby in the house is the difficulty in preventing the nurse from a surreptitious defrauding of the needy foster-child. Unless such a procedure can be absolutely controlled it is better that the nurse leave her own baby behind. There are instances, however, when the opportunity for nursing both chil-

dren is an advantage. This is especially true when the supply of milk is very abundant, since the foster-baby may be overfed, or if too weakly to empty the breast properly the secretion is not maintained and may dry up. The employment of a breast-pump, or expression of the milk, is by no means as satisfactory for this emptying as is the direct sucking by an infant. Whether or not the wet-nurse's child accompany her, it is only humane that the employer see to it that the infant is well cared for.

Selection of a Wet-nurse.—The wet-nurse selected should be strong, between 20 and 30 years of age, of quiet disposition, and not nervous. Other things being equal, her general health should be good, although delicate looking wet-nurses not infrequently supply excellent milk. Particularly she should be not anemic, not too fat, and free from any evidence of syphilis or tuberculosis, or of parasitic or infectious disease of the skin. The presence of a positive von Pirquet reaction is, of itself, not a contra-indication to her employment. The existence of apparent health in the mother, and especially in her own child makes it probable that syphilis is absent; but whenever possible a Wassermann test should be made. Conversely it may be said that a syphilitic foster-baby should not nurse from a healthy wet-nurse. The nipples must be prominent enough to be grasped easily by the child, and be free from fissures. The *mammæ* should be firm and hard before nursing, and should become flabby to some extent when emptied. If they do not, their size and shape before nursing probably depend on the fact that they contain more fat than glandular tissue. The best test, however, of the amount of secretion is the weighing of the infant before and after nursing, since the appearance of the breast is so often deceptive. The weighing should be done after each nursing for a day or more. A single weighing is not satisfactory. In connection with the weighing it may be said that this is a check, too, upon a young infant receiving far more milk than it can properly digest. (See Quantity of Milk, p. 92.)

The nurse's baby should by preference be some weeks old in order to show that the milk has been nutritious, and that a condition of equilibrium may have been reached. If her child is healthy and well nourished it is an indication that the milk will probably agree with the foster-child. There is no necessity of having the nurse's own child and her foster-child of very nearly the same age, inasmuch as there is comparatively little change in a woman's milk after the 1st month. (See p. 103.) Yet, on the other hand, the difference should not be too great, especially if her own child is much older, lest the quality deteriorate or the secretion cease before the younger foster-child is of the age to wean.

When practicable the nurse's milk should be analyzed before she is employed. Should her milk not seem sufficiently abundant during the first days after she is engaged, one must not at once despair of her, for the scantiness may be remediable by hygienic treatment, or may be the result merely of nervous impressions such as excitement from assuming the new position, worry at weaning her child, and the like.

The moral character of the wet-nurse has no effect whatever upon the child; that is to say, traits of character are not transmitted with the food. It is similarly a matter of indifference whether her color is white or black. Yet her character cannot be entirely disregarded for other reasons. If she is intemperate, vicious, and irresponsible, an infant cannot be safely entrusted to her charge, however normal her milk-secretion may be. A wet-nurse who is a primipara should certainly not be rejected because

she is unmarried. If she has had more than one illegitimate child she is probably depraved in other respects and should not be trusted.

Hygiene of the Wet-nurse.—The rules for the diet and hygiene of the wet-nurse are those already given for the nursing mother (p. 84). Yet it is important to remember that the diet of the household is probably not that to which the nurse has been accustomed, and that no sudden change to a richer and more delicate diet should be made, lest some alteration in the character or quantity of the milk due to indigestion or other causes result. It is important, too, that a woman who has followed an active working life, be not forced to sit about and do nothing. Such a course is sure to affect her milk. She should as far as possible do the kind of work, eat the sort of food, and in general live the life to which she has been accustomed.

HUMAN MILK

Quantity.—The quantity secreted depends largely upon the demands made by the infant. Consequently there is less milk supplied in the early period of infancy than later. The estimation of the average secretion can therefore be only approximate. Systematic studies extending over some months, made by weighing infants before and after each nursing, have been carried on by very few investigators. Feer¹ has combined a number previously published with experiments of his own. The following figures deduced from his table represent the average total daily amounts taken by the infants studied, as well as the average at each nursing during the 1st half year:

TABLE 27.—SECRETION OF BREAST MILK

Age	Average amount taken daily		Average amount at each nursing	
	Grams	Fluidounces	Grams	Fluidounces
1st week.....	310	10.5	66	2.2
2d week.....	558	18.9	90	3.0
3d week.....	601	20.3	97	3.3
4th week.....	666	22.5	111	3.7
5 to 8 weeks.....	725-818	24.5-27.6	125-141	4.2-4.8
9 to 12 weeks.....	800-832	27.0-28.1	138-146	4.7-4.9
13 to 16 weeks.....	847-879	28.6-29.7	154-157	5.2-5.3
17 to 20 weeks.....	842-922	28.5-31.2	153-174	5.2-5.9

During the first 2 days of the 1st week the amount taken by the child is necessarily much less than later, only 10 to 20 c.c. (0.34 to 0.68 fl.oz.), since secretion has not been fully established. The figures obtained by Selter² in 3 systematically studied cases are somewhat lower than those of Feer. All estimates made in this way, however, show only the amount of milk taken by the child, and not that which the breast is capable of secreting. An average breast should give an average secretion of 1200 grams (40.6 fl.oz.) a day during the nursing period (Schlossmann).³ A wet-nurse feeding several infants may not infrequently secrete daily in the neighborhood of 2 litres (67.6 fl.oz.), or occasionally even 3 litres

¹ Jahrb. f. Kinderh., 1896, XLII, 195; 1902, LVI, 421.

² Archiv f. Kinderheilk., 1903, XXXVII, 91.

³ Arch. f. Kinderh., 1902, XXXIII, 194.

(101.4 fl.oz.). Laurentius¹ observed an instance where 3450 grams (116.66 fl.oz.) were produced in a single day; and Rommel² the secretion of 4125 grams (139.48 fl.oz.) in one day.³

Colostrum.—The secretion occurring during the first 3 or 4 days after the birth of the child, and to some extent before its birth, is called "colostrum." It differs from milk not only to some degree in chemical composition, but in the presence of the "colostrum corpuscles." These are cells, probably, as Czerny⁴ believes, of lymphoid nature or are derived in part from the "mast" cells (Unger).⁵ They are 4 or 5 times larger than the average leucocyte, are phagocytic, contain a nucleus, are filled with fat-globules, and are at first very numerous (Fig. 16). They persist in constantly decreasing numbers until the end of the 2d week. A longer continuation indicates that the milk is not in a healthy state. The

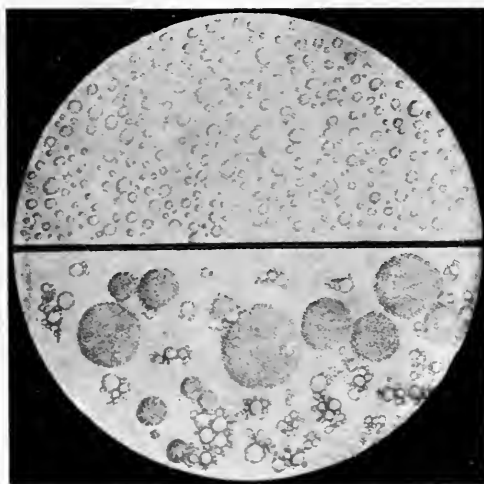


FIG. 16.—MILK AND COLOSTRUM.

Upper half human milk; lower colostrum. Magnified. (*Thiemic, Feer's Lehrbuch der Kinderheilkunde*, 1914, 6.)

corpuscles reappear also when nursing is intermitted for a day or two, or when the breasts are not sufficiently emptied. Any breast-milk in which colostrum-corpuscles are present in decided numbers is liable to disagree with the infant. In addition to the corpuscles and fat-globules, mononuclear and polymorphonuclear leucocytes may be found. The fat-globules of colostrum are very unequal in size.

Most investigators agree that the colostrum of the first few days before milk begins to be secreted is richer in protein and salts and poorer in sugar and fat than the fully developed milk. The results from analyses made by different investigators is shown in the following table:

¹ Arch. f. Kinderh., 1912, LV1, 275.

² Münch. med. Wochenschr., 1905, LI1, 444.

³ The calculations of the English measure as given above are on the assumption that grams were taken as the equivalent of cubic centimeters. If the milk was weighed, the volume secreted will be slightly less than the figures given.

⁴ Henoch's Festschrift, 1890, 194.

⁵ Virchow's Archiv., 1895, XLI, 159.

TABLE 28.—COMPOSITION OF COLOSTRUM

	Adrianne ¹	Woodward ²	Söldner ³	König ⁴
Fat.....	3.46	4.00	3.26	3.34
Protein.....	2.05	1.90	1.79	3.07
Lactose.....	6.35	6.50	5.83	5.27
Ash.....	0.23	0.20	0.30	0.40
Total solids.....	12.10	12.50	12.09	12.08
Water.....	87.89	87.50	87.91	86.70

The difference in these figures depends largely upon the material analyzed. The analysis of König is of the colostrum of the first 3 days; the others of that of the first 5 or 6 days or longer.

The color of colostrum is deep lemon-yellow due to the presence of the corpuscles, the reaction decidedly alkaline, and the specific gravity 1040 to 1060. It is coagulated firmly by heat and by acids. The excess of protein and of salts is probably the cause of the slightly laxative action which colostrum is believed to possess. The fat of colostrum is richer in oleic acid than is that of human milk (Engel);⁵ and according to Bauer⁶ the protein is of the nature of a direct transudate, in contradistinction to that of fully developed milk, and is analogous to that found in blood-serum. The mineral matter differs from that of milk in that magnesium and phosphorus are in relatively greater amounts and possibly the calcium also, while the proportion of sodium is smaller.

The chemical characteristics of colostrum persist to some extent, although in a constantly diminishing degree, during the first 2 weeks after childbirth, by which time the "colostrum period" may be said normally to have closed, and the "equilibrium milk" or "mature milk" is secreted.

Composition of Human Milk.—Human milk, like that of other animals, is an emulsion consisting chiefly of water, containing suspended or dissolved in it certain amounts of fat, sugar, protein and salts. It is the product of actual secretion of the breast, the epithelial cells not undergoing destruction in the process of its formation. It is not a transudate, although to a limited extent substances, such as certain drugs, may simply pass through the gland and be excreted in the milk. It begins to be secreted by about the 3d day after birth, or sometimes later, the characteristics of colostrum being still present to some extent. It is of a bluish-white color and has a slightly sweetish taste and an amphoteric or faintly alkaline reaction with litmus paper. Kerley, Gieschen and Myers⁷ have shown that with phenolphthalein it is always faintly acid. The white color is not due solely to the existence of fat in emulsion, but probably also to the presence of casein and calcium phosphate, since it is to be seen as well in fat-free milk. The specific gravity averages about 1030 to 1031 with a normal range of 1028 to 1034. Greater variations occur, and König⁸ gives a range of 1020 to 1036. Coagulation is caused by acid, but only to a slight degree as compared with cow's milk; and

¹ Arch. of Ped., 1897, XIV, 22.

² Journ. Exper. Med., 1897, March.

³ Zeit. f. Biol., 1898, XXXVI, 280.

⁴ Chemie des menschl. Nahrungsmittel, 1903, I, 100.

⁵ Sommerfeld's Handb. der Milchkunde, 1909, 810. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1915, 96.

⁶ Dtsch. med. Wochenschr., 1909, XXV, 1657.

⁷ New York Med. Rec., 1903, Aug. 8.

⁸ Loc. cit., 110.

that by rennet does not take place except in the presence of acid, and then less firmly than in the case of cow's milk. The casein is changed to paracasein under the influence of rennet. Under the microscope are found crowded oil-globules varying in size (Fig. 16). In addition to these are a limited number of leucocytes, and a very large number of extremely minute ultramicroscopic particles consisting of casein (Alexander and Bullowa).¹

Numerous and careful analyses of the composition of milk have been made. There is necessarily a considerable variation among individual women and those of different races and conditions of life, and even of the same woman at different times of the day. The following table, after Czerny and Keller,² shows some of the variations according to the analy-

TABLE 29

Investigators	Fat, per cent.	Sugar, per cent.	Protein, per cent.	Ash, per cent.	Solids, per cent.
Pfeiffer ³	0.76-9.05	4.22-7.65	1.049-3.04	0.104-0.446	8.23-15.56
Johannessen and Wang ⁴	2. 7-4.6	5. 9-7.8	0. 9-1.3		
V. and J. S. Adriance ⁵	1.31-7.61	5.35-7.95	0.23-2.60	0.09-0.28	9.19-15.31
Guirand ⁶	1.75-6.18	6. 7-7.7	0.85-1.4	0. 1-0.27	11. 2-16.3
Camerer and Söldner ⁷	1.27-5.77	5.35-7.52	0.83-1.87	0.11-0.36	9.41-14.11
Schlossmann ⁸	1.65-9.46	5.2-10.90	0.56-3.4		

ses of different investigators:

For an approximate analysis of average human milk the following may be assumed as a working guide:

TABLE 30.—AVERAGE COMPOSITION OF HUMAN MILK

Fat.....	3.5- 4.0 per cent.
Sugar.....	6.5- 7.0 per cent.
Protein.....	0.1- 1.5 per cent.
Ash.....	0.2 per cent.
Water.....	87.0-88.0 per cent.
Total solids.....	12.0-13.0 per cent.

There are also present in small amounts certain nitrogenous substances not of the protein class; citric acid; and a number of other bodies little understood.

THE DIFFERENT CONSTITUENTS OF MILK AND THEIR NORMAL VARIATIONS

Fat.—The fat in milk is in the form of many globules of size varying from 0.0033 to 0.01 mm. (0.00013 to 0.00039 inches) in diameter, and in number averaging 1,026,000 to the c.mm (Bouchut).⁹ These form an emulsion with the milk-plasma. It is disputed whether the globules are

¹ Journ. Amer. Med. Assoc., 1910, LV, 1196.

² Des Kindes Ernährung, Ernährungstörungen und Ernährungstherapie, 1906, I, 416.

³ Verhandl. XI, Versamml. d. Gesellsch. f. Kinderh., 1894, 131.

⁴ Zeit. f. physiol. Chem., 1898, XXIV, 499.

⁵ Arch. of Ped., 1897, XIV, 27.

⁶ Thèse de Bordeaux, 1897.

⁷ Zeit. f. Biol., 1898, XXXVI, 280.

⁸ Archiv f. Kinderh., 1900, XXX, 324.

⁹ Gaz. des hôpitaux, 1878, LI, 66; 75.

surrounded by a distinct albuminous membrane. There is clearly some form of coating which prevents their coalition one with another, but the exact nature of this is not known. Abderhalden and Völtz¹ maintain that it is not casein, and perhaps is a mixture of different proteins. Chemically the fat consists principally of the oleate, myristate, palmitate and stearate of glycerole, the first being especially abundant. The others, as well as the volatile acids—butyric, capric, caproic, and caprylic—are present in small amount as compared with cow's milk (Ruppel);² viz., 2.5 per cent. of the total fat in human milk; 27 per cent. in cow's milk (Morse and Talbot).³ The proportion of the total fat in the milk is capable of great variation, more than that of any other constituent. The percentages may certainly range from 2 up to 5 or even more in milk which may yet be called healthy for practical purposes. Not infrequently these limits are much exceeded, the percentage dropping occasionally to 1.5 or even less, while in one instance I observed it exceed 11 in milk otherwise normal.

Sugar.—The sugar present in human milk is identical with, or closely allied to, the lactose occurring in cow's milk. Its proportion is less liable to vary than that of fat and protein, and is fairly fixed at from 6 to 7 per cent. in healthy milk. Variations decidedly below or above these figures sometimes occur.

Protein.—The protein-constituent of human milk is far from being a simple one and its chemistry is not yet fully understood. The total protein-matter, as studied by later and more accurate methods, may be given as from 1 per cent. to 2 per cent. within entirely normal limits, and with a decided capability of variation from these figures. A percentage of 3.5 or even more is not uncommon, and is sometimes digested well, though oftener not. Two principal protein bodies are uniformly recognized: casein and lactalbumin. There exists in addition the protein substance frequently described by some writers as "lactoglobulin," and still others have been reported. Wroblewski⁴ describes one rich in sulphur which he called "opalisin." For practical purposes, however, only the first two need be considered. One of the principal characteristics of human milk as compared with cow's milk is the relatively large quantity of lactalbumin present, the relation which the amount bears to that of casein being, according to Lehmann⁵ as 5 to 12; albumin with lactoglobulin 0.5 per cent.; casein 1.2 per cent. König⁶ gives lactalbumin 1.21 per cent., casein 0.80 per cent., while in cow's milk, according to the same authority, the figures are 0.51 per cent. and 2.88 per cent. respectively. Ciccarelli⁷ found lactalbumin from 62.1 to 73.1 per cent. and casein from 26.9 to 37.9 per cent. of the total protein. The results obtained by different investigators are clearly by no means uniform, but all agree that in human milk the percentage of lactalbumin as compared with that of casein is greater than obtains in cow's milk. Average figures would probably give lactalbumin and globulin 39 to 44 per cent.; casein 41 per cent.; residual nitrogen 15 to 20 per cent. (Talbot).⁸

The casein is a nuclealbumin which is held in suspension, but not

¹ Zeit. f. phys. Chem., 1909, LIX, 13.

² Zeit. f. Biol., 1895, XXXI, 1.

³ Diseases of Nutrition and Infant Feeding, 1915, 107.

⁴ Zeit. f. phys. Chem., 1898-99, XXVI, 308.

⁵ Arch. f. d. gesamte Physiol., 1894, LVI, 577.

⁶ Chem. d. menschl. Nahrungs- u. Genussmittel, 1903, 110.

⁷ La Peliatria, 1908, VI, 12.

⁸ Amer. Jour. Dis. Child., 1911, VII, 445.

dissolved, in the milk-plasma. It is believed that it is not entirely identical with the casein of cow's milk, being not so readily coagulated by acids, salts or rennet, and the coagulum formed being fine and loose, and dissolving readily in an excess of acid. On the other hand, it has been claimed that this depends upon the diverse percentages present in the two forms of milk and the relative difference in the amounts of lactalbumin and of salts. Whatever the chemical differences may be, the work of Bordet,¹ Wassermann² and others, has shown from a biological standpoint that dissimilarities do exist between the caseins of different mammalian milks. The experiments have demonstrated, that whereas the blood-serum of animals sensitized to one mammalian milk will react with the protein of the milk of this species, it will not do so with other species.

The lactalbumin is in solution in the milk. It is allied to or identical with serum-albumin.

Mineral Matter.—The salts of milk are a somewhat complex substance. A study of those of human milk by Harrington and Kinnicutt³ gave the following results:

TABLE 31.—COMPOSITION OF THE MINERAL MATTER OF HUMAN MILK
(Harrington and Kinnicutt)

Calcium phosphate.....	23.87
Calcium silicate.....	1.27
Calcium sulphate.....	2.25
Calcium carbonate.....	2.85
Magnesium carbonate.....	3.77
Potassium carbonate.....	23.47
Potassium sulphate.....	8.33
Potassium chloride.....	12.05
Sodium chloride.....	21.77
Iron oxide and alumina.....	0.37
	<hr/> 100.00

Another reliable analysis by Söldner⁴ is as follows:

TABLE 32.—PERCENTAGES OF MINERAL MATTER IN HUMAN MILK
(Söldner)

Potassium oxide.....	0.0884
Sodium oxide.....	0.0357
Calcium oxide.....	0.0378
Magnesium oxide.....	0.0053
Ferric oxide.....	0.0002
Phosphoric oxide.....	0.0031
Sulphuric oxide.....	0.0090
Chlorine.....	0.0591

A series of analyses by Holt, Courtney and Fales⁵ of milk of the mid-
dle portion of lactation gave the following results:

TABLE 33.—PERCENTAGES OF MINERAL MATTER IN HUMAN MILK
(Holt, Courtney and Fales)

Calcium oxide.....	0.0458
Magnesium oxide.....	0.0074
Phosphoric oxide.....	0.0345
Sodium oxide.....	0.0132
Potassium oxide.....	0.0609
Chlorine.....	0.0358
Total ash.....	0.2069

¹ Ann. de l'Institut. Pasteur, 1899, XIII, 225.

² Verhandl. XVIII, Cong. inn. Med., 1900, 501.

³ Rotch, Pediatrics, 1901, 130.

⁴ Zeit. f. Biol., 1902, XLIV, 61.

⁵ Amer. Jour. Dis. Child., 1915, X, 229.

Probably the amounts of the ingredients vary much in different milks, and certainly in the fore-milk and the last milk of a single nursing. The total quantity of mineral matter in human milk is likewise subject to decided variation within normal limits, from 0.15 to 0.25 per cent. being a not unusual range. Human milk as compared with that of the cow is poorer in calcium and in phosphorus, but possesses a larger amount of iron. The phosphorus in human milk is to a great extent in a different state from that contained in cow's milk. It exists largely in organic combination, and is believed to be chiefly in the form of lecithin and nucleone, both of which are in small amount in cow's milk. About 77 per cent. of the phosphorus in human milk is in organic combination, against about 27 per cent. in cow's milk.

Citric Acid.—This is present in an average amount of 0.05 per cent. (Scheibe).¹

Ferments.—It seems probable from the investigations of later years that various ferments and allied bodies play a rôle of some importance in rendering human milk better suited than cow's milk for the needs of the infant's economy. This is, however, very uncertain, since cow's milk, at least, appears to be as digestible after boiling, which destroys the ferments, as before. Moreover the difficulty in keeping the milk from the action of bacteria renders the study very difficult, inasmuch as these may produce the same effect as do the ferments (Morse and Talbot).² A diastatic ferment, amylase, reported by Bechamp³ and others, is claimed not to be present in cow's milk. A fat-splitting ferment, lipase, is stated by Marfan and Gillet⁴ to be active in human milk, and but slightly so in cow's milk. One which decomposes salol is described by Nobécourt and Merklen⁵ as present in human milk, but absent from cow's milk. The existence of this ferment is questionable. Moro and Hamburger⁶ found one which coagulates fibrin, likewise absent from cow's milk. A proteolytic ferment is described by Babcock and Russell⁷ and others, and a glycolytic ferment by Spolverini⁸ as present in both kinds of milk. Superoxidase, peroxidase and reductase occur in both human and cow's milk.

Protective and Other Bodies.—Moro⁹ showed that although no bactericidal substances were to be found in human milk, yet that the blood-serum of breast-fed children exhibited a bactericidal power much greater than that of those artificially fed, and that the former do not contract pyogenic diseases so easily. The protective power would appear to be drawn from the mother's milk. Diphtheria antitoxin is found in the milk of immunized animals, and, indeed, the well-known comparative immunity of early infancy as regards many of the infectious diseases probably depends in part on the presence in the breast-milk of immunizing substances, and the passage of this into the serum of the child. Specific agglutinins are also probably transmitted to the child through the mother's milk.

¹ Ref. Morse and Talbot, *Diseases of Nutrition and Infant Feeding*, 1915, 110.

² *Loc. cit.*, 116.

³ *Compt. rend. de l'acad. des sci.*, 1883, XCVI, 1508.

⁴ Marfan, *l'Allaitement*, 1903, 31.

⁵ *Rev. mens. des malad. de l'enf.*, 1901, XIX, 138.

⁶ *Wien. klin. Wochenschr.*, 1902, No. 5, 121.

⁷ *Rep. Wisconsin Agric. Station*, 1898. Ref. Raudnitz, *Ergeb. d. Physiol.*, 1903, II, 1 Abt., 285.

⁸ *Archiv de méd. des enf.*, 1901, IV, 705.

⁹ *Jahrb. f. Kinderh.*, 1902, LV, 396.

Caloric Value.—According to the estimations of Schlossmann¹ the average value of a liter of human milk in large calories is 782 (740 calories per quart), while that of average cow's milk may be assumed as about 670 calories (654 per quart). Heubner² gives 700 calories (663 per quart) for human milk and 690 (653 per quart) for cow's milk. (See *Caloric Value of Cow's Milk*, p. 110.) The caloric value of the individual milk-elements is considered elsewhere (pp. 53 and 123).

Action of the Different Constituents of Human Milk.—(See also pp. 48 and 127.)

Water.—The large percentage of water in the milk indicates the need of the infant especially in that particular. Concentrated food is not as easily assimilated as a rule, even though the actual amount of solid matter ingested is not altered, and the work thrown upon the excretory organs is also increased if sufficient water is not ingested. The amount of water required by an infant is proportionately many times greater than in adult life. There is little need, however, of water given by itself when the diet is entirely liquid, since enough is obtained in the food; but for other reasons it is well to accustom an infant to it.

Fat.—One of the functions of the fat of the milk is that of increasing the weight of the body by the deposition of it in the tissues. It is also needed for the proper formation of certain tissues, notably of the nervous and osseous systems. A still more important function is the sustaining of the body-temperature. As already stated (p. 49) this object is gained by metabolic processes much more easily from the consumption of fat than from that of protein. This leaves the latter for the object for which it was primarily intended; *i.e.*, the formation of nitrogenous tissue. A deficiency in the amount of fat in the breast-milk requires that a very much larger amount of protein or of carbohydrate must be ingested than the needs of the child in other respects demand, or perhaps than its digestive powers will tolerate.

Another result of a proper amount of fat in the milk is a normal condition of the stools, which always contain a considerable quantity of it. Too little fat is liable to result in constipation. On the other hand, too high a percentage of fat is prone to produce disturbance of the gastric digestion and diarrhea, or sometimes constipation with soap-stools.

Sugar.—Like the fat the function of sugar is the production of heat needed by the child. To a considerable extent it may replace the fat, both for this purpose and for the formation of new fatty tissue. Yet an infant who must rely for any length of time upon sugar to make up for a deficiency of fat in the milk is liable to suffer eventually; for, even though gaining in weight, tissue-formation is prone to be defective, and the strength is below normal. What is said here of the sugar natural to human milk applies equally well in a large degree to other varieties, as well as to the starchy food used later in infancy, since this latter must be converted into sugar before it can be absorbed. The effect of other sugars and of starch upon digestion are considered more fully elsewhere. (See pp. 49 and 128.)

Protein.—The protein is the essential tissue-producing substance of the milk, since it is the only one containing nitrogen. Too low a percentage of protein is certain to be followed eventually by malnutrition in some form, even though the infant may show no wasting. Protein produces heat in its consumption. Consequently an infant could sustain life and weight for a time on a diet solely proteid in nature. To

¹ Arch. f. Kinderh., 1900, XXX, 324.

² Lehrb. d. Kinderh., 1911, 51.

accomplish this, however, so large an amount is required that the general condition of the child would almost certainly be harmfully affected in some way; and for the production of heat the other elements of the food should be relied upon. (See p. 50.)

Mineral Matter.—Although the entire purposes of the salts are not understood, yet they certainly play an important and essential part in nutrition. The salts of calcium are required for the formation of osseous tissue. In addition calcium in the milk is a requisite for the proper action of rennet. Phosphorus, too, is needed for the osseous and the nervous structures. The deficiency in the iron of the milk is made up by the withdrawal by the system of the iron stored in the liver.

Bacteria in Milk.—Healthy human milk generally contains a few bacteria. This has been shown by Honigmann,¹ Cohn and Neumann,² Ringel,³ Köstlin,⁴ and others. The germs make their way in through the nipple. Consequently the first milk drawn by the child when nursing is that in which they are contained. The remaining milk is generally sterile. The principal germs found are the staphylococcus albus and, less commonly, aureus (Köstlin). For practical purposes human milk may be considered germ-free. When the mammary gland is suppurating the number of germs in the milk is necessarily much increased. This may also sometimes be true where the woman is the subject of sepsis. Certain pathogenic germs, as those of tuberculosis and typhoid fever, may very exceptionally occur in the milk of women suffering from these diseases. Wang and Coonley⁵ in 450 examinations of the milk of 28 tuberculous women found tubercle bacilli in no instance with 1 possible exception. Uhlenhuth and Mulzer⁶ inoculating rabbits with milk from syphilitic women were able to produce in them the lesions of syphilis.

Characteristics and Effects of Poor Milk.—Owing either to the individual peculiarity of the baby, or to faults in the quality of the milk, some infants fail to digest the maternal secretion but thrive on that of a wet-nurse; or the milk which disagrees with one infant suits another perfectly. An analysis of the milk will often show in what the fault in composition consists. In other instances the milk is found to be chemically normal, yet does not agree. Much more important than the analysis is the character of the symptoms produced, and especially a failure of the normal gain in weight; and one can afford to temporize with moderate diarrhœa, vomiting, or colic, rather than wean the baby if the weight is satisfactory. Of course this does not apply if the symptoms are severe. One of the faults of the milk is that of being too rich in both fat and protein; the percentage of sugar not varying materially in the milk of different women. This over-rich milk is oftenest observed in the case of women who are taking too little exercise, eating too freely, and digesting well. The infant exhibits the various symptoms of indigestion and often loses weight. Submitting to a proper dietetic and hygienic regimen will usually correct the difficulty. Occasionally it is possible to relieve the infant's condition by allowing it to nurse for a few minutes only and then giving it a bottle of water, thus diluting the milk in the stomach. Less often the fat is increased and the protein normal in amount. Sour vomiting and curds in the stools are then liable to appear.

¹ Zeit. f. Hygiene u. Infectiouskrankh., 1893, XIV, 207.

² Virchow's Archiv., 1891, CXXVI, 391.

³ Münch. med. Woch., 1893, 513.

⁴ Arch. f. Gynäk., 1897, LIII, 201.

⁵ Jour. Amer. Med. Assoc., 1917, LXIX, 531.

⁶ Deut. med. Woch., 1913, XXXIX, 879.

Much more frequently, however, a poor milk is supplied containing either a low, or oftener a high percentage of protein and a very low percentage of fat. Poor milk of this sort is scanty in quantity, and is seen oftentimes in neurotic, debilitated, anemic, over-worked, or over-anxious women. The infants do not thrive and may be constipated and exhibit no symptoms of indigestion whatever, or may sometimes suffer (if the protein is in large amount) from intestinal indigestion with colic and loose stools, often brownish and offensive. Vomiting is not a frequent symptom. Efforts should, of course, be made to improve the quality of the milk (p. 106) by attending to the condition of the mother; but when the trouble continues after the early weeks of the child's life it is not likely that these efforts will be successful.

Examination of the Breast-milk.

There are several indications that milk is being secreted in sufficient *quantity*. If the breasts fill up well and become hard and round between the nursings; if the infant nurses from but one breast and is satisfied by that; and if it does not require more than 20 minutes to complete its nursing, the probability is that the milk secreted is abundant. On the other hand, if the breast remains soft and flabby; if the child nurses much too long and seems dissatisfied, or will nurse but a few minutes and then refuse to make further efforts; if it cries much, yet seems to have no colic or other disease; and if it does not gain in weight, yet has no illness to account for this, the milk is probably insufficient in quantity or perhaps poor in quality.

A direct method is to pump out or press out the breast-milk and measure it. Yet this is often unsatisfactory owing to the frequently experienced difficulty in emptying the breast in this way. A rough estimation of the character and quantity of the milk may be made by seeing with what degree of ease and in what quantity it can be expressed from the breast by the hand. The "thumb-nail test" also is serviceable to a limited extent, a large drop—not, however, the first which comes from the breast—being drawn upon the thumb nail, and its color, transparency and coherence indicating the richness of the milk. Normal milk should be white and opaque, and should preserve the drop-form even when the nail is held vertically. All this, however, is but approximate. The only satisfactory method of determining what amount of milk the child really obtains is by weighing it or the mother immediately before and immediately after the nursing, using scales which record ounces.

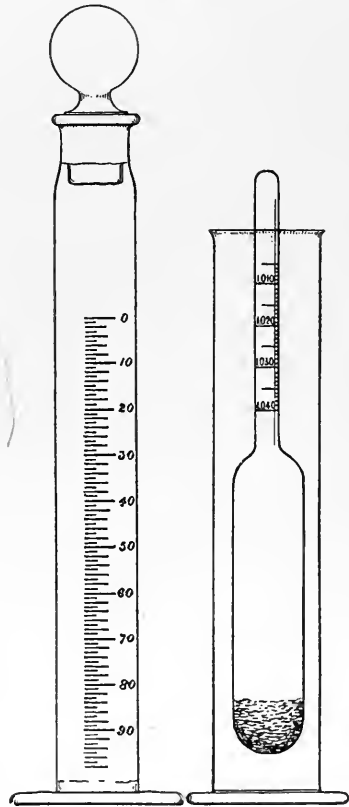


FIG. 17.—CREAM GAUGE AND LACTOMETER.

For approximate analysis of human milk. (*Holt, Arch. of Pediat.*, 1893, X, 193; 202.)

As regards the examination of the milk in other respects, the *reaction* is tested by litmus paper, or still more accurately by a solution of phenolphthalein. The *specific gravity* is determined by any small specific gravity glass which does not require much milk. A small urinometer (Fig. 17) answers every purpose. The accurate scientific estimation of the *percentages* of the milk-elements, with the exception of the fat, can be made only by one having a considerable knowledge of chemistry. Various methods may be employed, however, which give satisfactory approximate results. The milk to be examined should preferably be all that can be pumped or expressed from the breast. A less accurate procedure is to allow the infant to nurse for a moment, wash the nipple, and then pump out a small portion of the middle milk. This should be kept on ice in a sterilized, well-corked bottle, and a similar quantity of several different nursings added to it during the day.

Inasmuch as the percentages of sugar and of mineral matter are so nearly constant, it is only the variation in the fat and protein which we need determine for practical purposes. Holt¹ devised a simple method for the approximate examination of human milk. The principle of this depends upon the mutual relationships of the fat, the protein and the specific gravity. The latter is first determined in the way explained. The percentage of fat is then estimated by a small creamometer. This is a cylindrical vessel (Fig. 17) holding 10 c.c. (0.34 fl.oz.) and graduated to show the percentage of cream present. It is filled with milk to the zero mark, corked well and allowed to stand at the room-temperature of about 70°F. (21.1° C.) for 24 hours. The percentage of cream is then read off and that of fat calculated on the basis that this will be to that of the cream as 3 is to 5; *i.e.*, 5 per cent. of cream equals 3 per cent. of fat. The average amount of cream should be 7 per cent. Having determined in this way the fat-percentage and the specific gravity, the other factor, the amount of protein, can be readily estimated by the following rule of proportion; *viz.* that the fat varies inversely and the protein directly with the specific gravity; that is to say, the higher the fat-percentage, the lower will be the specific gravity, and the higher the protein-percentage, the higher will be the specific gravity. For instance, if the fat is normal, a high specific gravity must be due to a high protein-percentage. If the specific gravity is high and the fat low, the protein may be assumed as normal, since the low fat-percentage accounts for the high specific gravity. Or, again, if the specific gravity is normal and the fat low, the protein must be low, otherwise the low fat-percentage would have increased the specific gravity above normal. The following table shows in a convenient manner the various relations which may be present:

TABLE 34.—METHOD FOR APPROXIMATE ANALYSIS OF HUMAN MILK

	Specific gravity, 70° F.	Cream, 24 hr.	Protein, estimated
Average.....	1.031	7%	1.25%
Normal variations.....	1.028-1.029	8%-12%	Normal (rich milk)
Normal variations.....	1.032	5%-6%	Normal (fair milk)
Abnormal variations... Low (below 1.028)		High (above 10%)	Normal or slightly below
Abnormal variations... Low (below 1.028)		Low (below 5%)	Very low (very poor milk)
Abnormal variations... High (above 1.032)		High	Very high (very rich milk)
Abnormal variations... High (above 1.032)		Low	Normal (or nearly so)

¹ Arch. of Ped., 1893, X, 193. Diseases of Children, 1916, 141.

The method detailed is intended to be only an approximate one, and the fat and protein of the milk may be determined with sufficient accuracy by other procedures which are but little more difficult and are quite within the power of one who is not a chemist. For the estimation of the fat a serviceable instrument is the Babcock centrifuge, which, however, requires 17.6 c.c. (0.598 fl.oz.) of milk (Fig 18). A similar apparatus needing but 5 c.c. (0.169 fl.oz.) (Fig. 19) is the milk-testing glass supplied for this purpose with the small Bausch and Lomb centrifugal machine designed for centrifugal analysis, and employing the solutions recommended by Leffmann and Beam.

The principle consists in the oxidation of the sugar and protein by sulphuric acid, after the addition of fusel oil and hydrochloric acid. After centrifugation for a few minutes the fat rises into the neck of the tube and the percentage may be read.¹

The percentage of protein may be determined directly with reasonable accuracy by the method described by Boggs.² This consists in precipitating it with phosphotungstic and hydrochloric acids in an Esbach tube and reading off the percentage after 24 hours.

Conditions Altering the Character of the Milk.—Some of the abnormalities in composition already described (p. 100) depend upon various known causes, among which may be enumerated the following:

(a) **Period of Lactation.**—This exercises some influence upon the composition of the milk; yet, as a rule, not to any very considerable extent. The statistics of different investigators are not entirely in accord.

The colostrum of the first few days, as we have seen, is rich in protein and in salts (p. 93). This is modified rapidly after the 3d day, and by the end of 2 weeks an average milk is obtained which varies but little, although individual milks may change considerably and irregularly from time to time. According to investigations made by Camerer and Söldner³ (57 analyses), V. and J. S. Adriance⁴ (120 analyses), and Schlossmann⁵ (218 analyses), there occurs a slight but steady diminution in the percentage of protein beginning 2 to 4 weeks after child-birth, the loss by the 7th month of lactation being about 0.5 per cent. Toward the end of lactation there is a decided

diminution in the amount of protein, the percentage of sugar slightly increases, while the mineral matter diminishes. The percentage of fat shows no regular change, unless it may be a slight decrease (Camerer and Söldner). Sharpless and Darling⁶ find no noteworthy alteration in any of the constituents which can be called characteristic of different periods of lactation.



FIG. 19.—
FLASK FOR
BAUSCH AND
LOMB CENTRI-
FUGE.

For determin-
ing the fat-per-
centage of milk.



FIG. 18.—
BABCOCK'S
MILK-TEST
BOTTLE.

(D. H. Burrell
and Co.)

¹ See Formulæ supplied by the Bausch and Lomb Optical Co. with the instrument.

² Johns. Hopk. Hosp. Bull., 1906, XVI, 1342.

³ Zeit. f. Biol., 1898, XXXVI, 277.

⁴ Arch. of Ped., 1897, XIV, 22.

⁵ Arch. f. Kinderh., 1900, XXX, 288.

⁶ Boston Medical and Surg. Journ., 1903, CXLVIII, 416.

(b) **Intervals of Nursing and Time at the Breast.**—Peligot¹ has shown by experiments with asses' milk that the longer the fluid is in the udder; *i.e.*, the longer the interval between nursings; the more watery it becomes. The same is true of human milk. It is apparently especially the fat which is increased in amount by shortening the nursing intervals. There is a difference, too, in the milk from the breast at the beginning, in the middle, and at the end of nursing, again the change being chiefly in the fat. The analyses made by Mendes de Leon² and by Adriance³ give figures illustrating the truth of this. Harrington's⁴ analysis of cow's milk tends

TABLE 35.—FAT-CONTENT OF HUMAN MILK

	Mendes de Leon Fat, per cent.	Adriance Fat, per cent.
Full breast.....	2.62	2.27
Middle milk.....	4.06	2.79
Nearly empty breast.....	6.02	3.65

to confirm this difference for human milk since similar conditions would naturally exist.

TABLE 36.—FAT-CONTENT OF COW'S MILK
(Harrington)

	Fat, per cent.	Total solids, per cent.	Water, per cent.	Ash, per cent.
Fore milk.....	3.88	13.34	86.66	0.85
Middle milk.....	6.74	15.40	84.60	0.81
Strippings.....	8.12	17.13	82.87	0.82

(c) **Number of Pregnancies and Age of Mother.**—According to Adriance⁵ the milk of women between 20 and 30 years of age is slightly richer in fat and protein and slightly poorer in sugar than that of older women. The same is true of primiparæ as compared with multiparæ. The differences, however, are neither very material nor very constant, and may be disregarded provided only that the health of the mother is good.

(d) **Pregnancy.**—This generally diminishes the quantity of the milk secreted and affects its quality, the amount of fat especially being reduced, and the milk sometimes approaching the character of colostrum-milk. In other cases the milk remains abundant and of good quality, and the infant could safely be kept upon the breast; but this is not the rule, and on the mother's account the child should be weaned.

(e) **Menstruation.**—No certain alteration of the milk follows if menstruation reappears. In some cases the percentage of fat is diminished and that of the protein and of the sugar increased or the general health of the infant may exceptionally be acutely disturbed. In most instances the character of the milk is entirely unaffected, or, perhaps, at most only during the days when the menses are present. Often the menses appear but once, and do not return until lactation is normally over. The

¹ Hermann, Handb. d. Physiol., V, Th. 1, 404. Ref. Rotch, Keating's Cyclop. Dis. Child., 1889, 1, 289.

² Zeit. f. Biol., 1881, XVII, 501.

³ Loc. cit., 22.

⁴ Rotch, Pediatrics, 1901, 133.

⁵ Loc. cit., 22.

greatest caution, therefore, should be exercised lest weaning be hasty and unnecessary. If acute symptoms develop the child could be artificially fed for the few days while menstruation lasts. An exhaustive study by Bamberg¹ led to the conclusion that there is no reason to believe that menstruation makes the milk harmful to the infant. (See also Causes making Nursing Inadvisable, p. 86.)

(f) **Diet.**—Diet often exercises great influence upon the character of the milk secreted. This is, however, chiefly upon the protein and the fat, the sugar and salts being little affected. Insufficient nourishment of the mother produces milk deficient in total solids, especially the fat. The protein is either diminished or increased in quantity. So, too, an excessive amount of liquid taken often diminishes the total solids in all particulars, although in other cases the amount of milk is much increased without any diminution in the percentage of the solid ingredients. An abundant and rich diet, if well digested, given to women previously under-fed, increases both the fat and the protein and the quantity of milk. On the other hand, an over-abundant diet sometimes tends either merely to increase the adipose of the mother, or to injure the quality of the milk secreted and produce indigestion. It has been thought that the percentages of fat and of protein are increased by a highly nitrogenous diet, but diminished by a vegetable one, and that a diet rich in digestible fat also increases the fat in the milk. It is questionable, however, whether any such alteration in diet affects any of the milk-elements except in the case of women who have been previously long under-fed. It is possible that certain vegetables which possess a strong odor and taste may communicate these to the mother's milk, which may thus become unpleasant to or disagree with the child. It is likely, too, that some women exhibit idiosyncrasies toward certain articles of food, and that after they have partaken of them their milk may disagree with the baby. These are, however, all exceptions, and as a rule a healthy nursing woman may eat any digestible food without fear of any special influence of it upon the child.

(g) **Exercise.**—This, too, influences the character of the milk decidedly. Deficient exercise combined with a healthy appetite is liable to increase the percentages of protein and of fat; a proper amount of exercise to decrease an excess, if the woman has been too inactive; excessive exercise, with constant fatigue, to increase the protein.

(h) **Mental and Nervous Influences.**—The affect of these is greater than perhaps from any other cause. It not infrequently happens that fright, anger, sorrow, or other great emotion will quickly so affect the milk that the infant is made ill by it. What the changes are is not certainly known. In some instances there is a sudden great diminution in the amount of the total solids, especially the fat, and increase in that of water. In others the protein is much increased in amount. In some cases there seem to be actual toxic changes produced in the milk. It sometimes even happens that the secretion of milk is quickly and permanently arrested by some emotional cause, or may have its quality affected and its amount much diminished by more prolonged nervous influences such as worry, repeated fatigue, and the like. The nervous mother is the one particularly liable to have a milk poor in fat and too rich in protein.

(i) **Illnesses.**—A good state of the general health is often a prerequisite to the secretion of good and abundant milk. In other cases

¹Zeit. f. Kinderh., Orig., 1913, VI, 424.

the milk continues unaffected in spite of the mother being delicate and frail. This is often at the expense of the mother. Acute temporary ailments have but little effect upon the milk. In more severe febrile diseases the amount of milk may be much diminished and the percentage of fat decreased and that of the protein increased.

(j) **Drugs.**—These may be divided into those which influence the secretion of milk and those which are excreted by the mammary glands with the milk. The former will be referred to again in considering the modification of breast-milk (see below). Of the latter it may be said that, contrary to the opinion among the laity, few drugs pass into the milk to any considerable extent, and the occurrence is so irregular and uncertain that efforts at medication of the child by way of the breast-milk would be unsatisfactory. The statements of investigators are largely contradictory. The following conclusions are based chiefly upon the publications of Fehling,¹ Houselot,² Engel,³ and others.

Alcohol imbibed in very large amounts may appear only in traces in the milk. It may, however, taken in this way by the mother or wet-nurse produce serious illness in the infant. *Atropine*, *hyoscine* and *colchicine* pass directly into cow's milk in small amounts, and probably do so in human milk as well. *Opium* taken by the mother very rarely has any effect on the child. Yet, as cases are on record where dangerous action has occurred, it is best to administer it cautiously and in doses smaller than usual. *Iodine*, given to the mother in the form of iodides or applied as iodoform in a dressing, passes readily into the milk. *Bromine* does so to some extent (Rosenhaupt)⁴ and occasionally in a way to affect the infant seriously. *Mercury* has practically no effect. It passes to the child only in small amount and after prolonged administration to the mother. *Chloral* has, as a rule, no influence. Any effect appears only in weakly children who are nursed within an hour after the ingestion of the drug by the mother. *Arsenic*, *antimony*, *hexamethylenamine*, *antipyrine*, and *phenacetin* pass into the milk to a limited extent. In the form of *arsphenamine* arsenic appears to have a decided effect on the child in some instances. *Quinine* has little if any influence on the infant as a rule, and only when given to the mother on an empty stomach, and when her child is still in its 1st half-year. *Lead* and *iron* enter the milk to some extent. *Salicylic acid* in its different combinations affects it very decidedly. The *mineral and vegetable acids* have no effect upon it. The *saline purgatives* occasionally pass into it to some extent, while the *vegetable purgatives*, as senna, aloes, rhubarb, and cascara, as a rule do not (Gow).⁵ *Chloroform* and *ether* appear to be without influence, with rare exceptions.

Modification of Breast-milk.—From what has been stated in previous sections it is evident that whereas the mother's milk may often be found insufficient or insuitable for the infant, it may sometimes be possible in various ways to modify the secretion in quantity or quality and thus to avoid the abandoning of nursing.

Quantity.—A threatened drying up of the secretion may often be prevented by seeing that the breast is thoroughly emptied at each nursing, giving both breasts if the milk is scanty. There is no better stimulant

¹ Arch. f. Gynäk., 1886, XXVII, 331.

² Rev. prat. d'obstet. et de pædiat., 1901, Nos. 1 and 2.

³ Sommerfeld's Handb. d. Milchkunde, 1909, 810. Ref. Morse and Talbot, Diseases of Nutrition and Infant Feeding, 1915, 114.

⁴ Arch. f. Kinderheilk., 1904, XL, 131.

⁵ Practitioner, 1893, L, 168.

to increase secretion than this. The taking of an abundant amount of liquid in some form is often an aid, with due caution that this does not merely increase the amount while diminishing the total solids in a breast-milk already poor in quality. Milk and cocoa, however, have no advantage over weak tea or water, so far as quantity is concerned; and in the case of a woman already well-nourished, may merely result in making her unduly fat. Malt liquors undoubtedly have a decided influence in increasing the quantity in some cases and may be used for this purpose, with caution that no addiction to alcohol is established. (See also p. 84.) In all under-nourished women a generous diet should be given and over-work avoided. In women of a nervous temperament the life should be as quiet as possible, all exciting influences avoided, and rest at night be undisturbed, the care of the infant at this time being delegated to the nurse, and, if it seems best, the night-feedings being of artificial food. With all nursing-women care should be taken that they spend a considerable part of each day quietly in the open air, driving, sitting, or taking easy walks. Among other methods which are recommended to increase the secretion are gentle daily massage of the breasts, faradization, and the administration of galactagogues. Regarding the last there is no certain evidence that any of these are of value. Experiments upon animals by some investigators appear to show such power in the administration of corpus luteum, placenta, the posterior lobe of the pituitary body, and the suprarenal glands, or in the injection of milk, but these have not been confirmed. Pilocarpine, cotton-seed, anise, and other drugs have been tried, but there is little evidence in their favor.

Quality.—In cases where the milk is in general too rich, the condition can often be remedied by insisting upon a more active life with abundant exercise, and by reducing the amount of food taken. Lengthening the interval between the nursings and diminishing the total solids ingested may be of avail. The excess of *protein* so common in defective breast-milk in neurotic women can sometimes be remedied by increasing the amount of exercise up to a healthful feeling of fatigue, and by taking measures to relieve the nervous condition. If, however, the exercise goes beyond this point, the percentage of protein may be increased. This is the condition of the milk existing in the over-worked and under-fed women of the poorer classes. Here an increase of diet and of rest may bring the milk closer to the normal. Influencing the protein-percentage by the diet is, however, usually unsuccessful except in those women who are over-fed and idle; and in the starvation cases in which, as sometimes happens, all the elements of the milk are deficient in quantity, instead of the protein being high as is usually the case. An excessive percentage of *fat* can be lessened, with the protein, in the over-fed and idle women referred to, by reducing the total amount of food ingested and increasing the exercise. It is also reduced by lengthening the nursing intervals. When the amount of fat is deficient little can usually be accomplished, except in the starvation cases, where an abundant diet and a diminution of work may increase the fat-percentage with that of the protein and the total secretion. Alteration of the diet has little effect on the secretion of fat in women already well nourished and exercising sufficiently. (See also p. 105.) The amount of *sugar* in the milk cannot be influenced with any certainty. It is less variable than either the fat or the protein. In some cases the administration of sugar has increased the percentage (Lust),¹ in others not at all.

¹ Monatssehr. f. Kinderh., Orig., 1912, XI, 236.

CHAPTER IV

ARTIFICIAL FEEDING IN THE FIRST YEAR

The nourishment of an infant with anything other than the secretion of the human breast is properly termed "artificial feeding, or "substitute feeding." The problem is often one of the most difficult which the physician encounters. The fact that we are dealing with a diet other than the natural one renders an absolute solution of it impossible, since, as far as our present knowledge extends, we are unable in any way to construct a food which is exactly like human milk. Moreover, we have to do always with the child as an individual, to whom general rules can apply only in a general way. Our inability to produce a food for infants which can be looked upon in any way as a standard must make us cautious in forming an arbitrary or hasty decision regarding the propriety or impropriety of a certain method of feeding in a given case, providing the infant is actually, and not only apparently, thriving upon its food. Nothing of this militates against the effort at an establishment of a scientific basis for the feeding of infants in general.

COW'S MILK

Of all foods the milk of some mammal is to be preferred as the basis, since it is in this class that the natural food of the infant belongs. That of various animals has at times been employed in different countries and for various theoretical reasons, the ass, goat, sheep, buffalo, sow, and mare having all been tried. These mammalian milks differ from human milk in various particulars, as is shown by the following table:

TABLE 37.—COMPOSITION OF MAMMALIAN MILK
(Richmond)¹

	Water, per cent.	Fat, per cent.	Sugar, per cent.	Casein, per cent.	Albumin, per cent.	Ash, per cent.
Cow.....	87.10	3.90	4.75	3.00	0.40	0.75
Goat.....	86.04	4.63	4.22	3.49	0.86	0.76
Ewe.....	79.46	8.63	4.28	5.23	1.45	0.97
Buffalo.....	82.63	7.61	4.72	3.54	0.60	0.90
Woman.....	88.2	3.3	6.8	1.0	0.5	0.2
Mare.....	89.80	1.17	6.89	1.84		0.30
Ass.....	90.12	1.26	6.50	1.32	0.34	0.46
Mule.....	91.50	1.59	4.80	1.64		0.38
Bitch.....	75.44	9.57	3.09	6.10	5.05	0.73
Cat.....	81.63	3.33	4.91	3.12	5.96	0.58
Rabbit.....	69.50	10.45	1.95	15.54		2.56
Llama.....	86.55	3.15	5.60	3.00	0.90	0.80
Camel.....	86.57	3.07	5.59	4.00		0.77
Elephant.....	67.85	19.57	8.84	3.09		0.65
Sow.....	84.04	4.55	3.13	7.23		1.05
Porpoise.....	41.11	48.50	1.33	11.19		0.57
Whale.....	48.67	43.67		7.11		0.46

¹ Dairy Chemistry, 1899, 323.

For practical purposes the milk of the cow is the only one which can generally be made use of in civilized countries, and any greater nearness in composition of other milks to human milk is of no special advantage, since all require *modification*, as by dilution or the addition of sugar, cream, water, etc., or in other ways, before they should be used.

Goat's Milk.—The milk of the goat may sometimes be advantageously employed when that of a cow is not obtainable. It is sufficiently similar to cow's milk to permit of it being modified in the same way. It is supposed to be superior in that the goat is comparatively free from tuberculosis; but the casein coagulates very firmly, and the milk acquires the unpleasant odor of the animal unless great precautions are taken. It is claimed to contain a greater percentage of iron than is present in cow's milk, and to be more like human milk in this respect (McLean).¹ It offers, however, no special advantage over the milk of the cow.

Characteristics of Cow's Milk.—The color of cow's milk is more yellowish-white than that of human milk. When absolutely fresh and obtained with proper care it possesses an indefinite, slightly sweetish taste and is practically without odor. The specific gravity ranges within normal limits from 1028 to 1033, with an average of 1032. The reaction as ordinarily obtained is faintly acid or amphoteric to litmus. With phenolphthalein it is always acid, and to a greater degree than human milk. The fat-globules vary greatly in size, the larger ones decidedly predominating. They are less numerous than in human milk and the emulsion consequently not so fine. Coagulation occurs readily by acid and by rennin, the resulting curd being firm and tough. When a certain degree of acidity is reached the milk will coagulate when heated. The nature of the process of coagulation by rennin has been much discussed. According to Bosworth and Van Slyke² the "casein" exists in milk in the form of a calcium caseinate. In the presence of rennin the molecule of this salt is broken up into two molecules of calcium paracaseinate. This being less soluble than the other, especially in the presence of a soluble calcium salt, it is precipitated as the curd. The addition of lime-water to milk forms a basic calcium caseinate which is not acted upon by rennin, the milk being alkaline and the coagulation not taking place by this ferment in an alkaline medium. When citrate of soda is added to milk, coagulation is likewise delayed, or finally inhibited if sufficient is used, on account of the formation of a soluble calcium-sodium caseinate. Rennin transforms this into calcium-sodium paracaseinate which is soluble. The presence of acid favors the action of rennin, perhaps by the formation of soluble calcium salts from the insoluble calcium phosphate present in the milk.

Effect of Heat on Milk.—Raising the milk to a temperature of 50°C. (122°F.) produces a slight skin upon the surface, which becomes greater if the milk be boiled. This is due largely to a partial coagulation and drying of the casein, and fat also in considerable quantity is contained in it. A temperature of 75°C. (167°F.) partially precipitates the lactalbumin, and prolonged boiling does this entirely. Heating up to or near the boiling point gives rise to the characteristic odor of boiled milk, partly by the production of a sulphur-compound from the lactalbumin, and a brownish tint is developed by the boiling if prolonged, dependent upon the caramelization of the sugar. The cream, too, rises imperfectly

¹ Zeit. f. Kinderh., Orig., 1912, IV, 168.

² Amer. Jour. Dis. Child., 1914, VII, 298; Journ. of Biol. Chem., 1913, XIV, 203.

or not at all in milk which has been heated for half an hour to a temperature of 65°C. (149°F.) (Rosenau),¹ owing to the effect upon the emulsion of the fat globules; and the action of rennin is interfered with. Even at a temperature of 145°F. (63°C.) the rising of the cream is prevented to some extent. Among other changes produced by heat are the driving off of the carbonic dioxide, oxygen, and nitrogen; a precipitation of the phosphates of the alkaline earths and of part of the citric acid; a diminution in the organic phosphorus, and a destruction in large part of the ferments, the alexins and the bactericidal properties of the milk. Few of these changes, however, occur except by a raising of the temperature to over 65°C. (149°F.) (Hippius);² and even the lactalbumin is not greatly affected at not over this temperature (Rupp).³ A continued elevation to this degree consequently leaves the milk practically unchanged in most respects, with the exception of the interference with the rising of the cream, which is, as stated, unsatisfactory and uncertain. A large number of bacteria of different sorts are destroyed by a temperature of 65°C. or even 60°C. (149°F. or 140°F.), although the peptonizing bacteria of the spore-bearing class and many acid-producing germs are more resistant. (See *Bacteria of Cow's Milk*, p. 113.)

Effect of Freezing.—Many varying statements have been made as regards the effect of freezing upon milk. It would appear to produce a separation of the oil-globules, but other action is uncertain. Long-continued freezing has been claimed to have a very decided influence upon the chemical composition (Pennington, *et al.*).⁴ Such freezing as may ordinarily be encountered in the household employment of milk does not usually have any harmful effect upon infants receiving it, although sometimes constipation, vomiting or diarrhea may be produced.

Caloric Value.—This naturally varies greatly with the differences in the composition of cow's milk. The range of figures is from 614 to 724 calories per litre (581 to 685 cal. per qt.). Heubner⁵ uses 690 to express the caloric value of a litre of milk (653 cal. per qt.).

Examination of Cow's Milk.—There is so little variation in the amount of protein, sugar and salts in mixed herd-milk that for practical purposes these may be taken as fixed amounts, and only the specific gravity, the reaction and the variation of the fat tested for, as well as the presence of bacteria and adulterants. Variations in the specific gravity may indicate that the milk has been tampered with. The removal of cream from the milk increases the specific gravity and the addition of water lowers it. The reaction is tested by litmus paper in the ordinary way. Strongly alkaline milk has probably been adulterated.

The amount of fat may be roughly estimated by the use of the cream-gauge, but only if the test is commenced before cooling after milking has taken place. A larger cylindrical gauge should be used than for human milk, and the calculation is made on the basis that the percentage of cream which has risen after 6 to 8 hours divided by 3 will give the percentage of fat present in the milk (Richmond).⁶ Cream rises more or less irregularly, and the best method for determining the fat is the use

¹ Hygienic Laboratory Bull., 1912, No. 56, 646.

² Jahrb. f. Kinderh., 1905, LXI, 365.

³ U. S. Dept. Agricult. Bull., 166. Ref., Grimmer, Monatsschr. f. Kinderh., Referat., 1912, XII, 670.

⁴ Journ. of Biol. Chem., 1908, IV, 353; 1913, XVI, 331.

⁵ Lehrb. d. Kinderh., 1911, I, 51.

⁶ Dairy Chemistry, 1899, 103.

of the Babcock centrifuge, or the smaller Leffman-Beam glass as described under Human Milk (p. 103). In the testing of cream with this it is necessary to dilute 5 or more times.

If it is desired to make an approximate estimation of the protein present, the method of Van Slyke and Bosworth¹ may be employed.

In addition it is sometimes necessary to examine milk for the presence of preservatives, especially boric acid, salicylic acid and formaldehyde, and to see whether it has been heated. The methods are not difficult, but are better described in special contributions to this subject. (See Richmond,² Freeman,³ Douglass,⁴ Whitefield,⁵ Winslow,⁶ and others.) A microscopical examination is also required in many instances for the detection of pus.

Composition of Cow's Milk.—The milk of healthy cows may vary widely, depending upon peculiarities of the individual and of the breed, the character of the food, the period of lactation, the amount of milk which has been drawn from the udder, the time of day, and other factors. Even the mixed milk of a herd is subject to changes, and different breeds vary one from the other. The variation is particularly evident in the fat, that of the other ingredients, especially the sugar and mineral matter being but slight. As a result of the conditions mentioned the published analyses of the chemical composition of cow's milk vary considerably. The following table gives approximate average analysis:

TABLE 38.—AVERAGE COMPOSITION OF COW'S MILK

Water.....	86.0–88.0 per cent.
Total Solids.....	12.0–14.0 per cent. ¹
Fat.....	3.5– 4.0 per cent.
Sugar.....	4.5 per cent.
Protein.....	3.5– 4.0 per cent.
Salts.....	0.7 per cent.

Nature and Normal Variation of the Constituents of Cow's Milk. (See also Human Milk, p. 95, for fuller description of the differences.)

Fat.—The fat, as stated, is by far the most variable element. Percentages run in round numbers all the way from 2 to over 6 per cent. in different cows and from 2.5 to over 5 per cent. in different herds (König).⁷ The herd-milk of Jersey and Guernsey cattle is from 1 to 1.5 per cent. richer in fat than average cow's milk; while the sugar and protein are also somewhat in excess. Holsteins and Ayershires produce a milk slightly poorer than the average in fat (Lythgoe).⁸ The amount of fat depends not only upon the breed, but upon the food given and the general treatment and health of the animal. It much resembles in composition the fat of human milk, olein and palmitin predominating; but the volatile fatty acids are relatively more abundant and the amount of oleic acid less than in human milk. The envelope of the fat-globules is similar to that of human milk.

Sugar.—The sugar may range in individual cows from 3.5 to 5.5 per cent. in round numbers, but in mixed herd-milk the variation is incon-

¹ New York Med. Jour., 1909, XC, 542.

² *Loc. cit.*

³ Med. Rec., 1899, Jan. 21.

⁴ Lancet, 1903, July 4.

⁵ Pediatrics, 1900, Jan. 1.

⁶ The Production and Handling of Clean Milk, 1909.

⁷ Chemie der menschlichen Nahrungs- u. Genussmittel, 1903, I, 119; 130.

⁸ Journ. Indust. Eng. Chem., 1914, VI, 899.

siderable. The nature of the food has some influence upon it. The sugar is lactose and generally believed to be identical with or closely allied to that of human milk.

Protein.—The protein may range from 2.5 to 4.5 per cent. (König) in different healthy cows, but in mixed herd-milk is fairly uniform, the variation being not over 0.5 per cent. The percentage of 4, formerly commonly accepted, is generally not equalled. That of 3.50 more fairly represents the standard. The protein consists of casein, lactalbumin, and small amounts of lactoglobulin, as in human milk but in different proportions. The results obtained by investigators vary, Lehmann¹ giving the ratio of the casein to the lactalbumin (with lactoglobulin) as 10:1 (casein 3 per cent., albumin 0.3 per cent.); Richmond² as 7.5:1 (casein 3 per cent., albumin 0.4 per cent.); Schlossmann³ as 6:1 (casein 3.19 per cent., albumin 0.53 per cent.); König⁴ as 5.5:1 (casein 2.88 per cent., albumin 0.51 per cent.); Van Slyke⁵ as about 4:1 (casein 2.48 per cent., albumin 0.66 per cent.) and White and Ladd⁶ as 3:1 (casein 2.94 per cent., albumin 0.9 per cent.). The casein coagulates more readily by acids and by rennin than does that of human milk, very possibly differs chemically from it, and certainly does so biologically. (See pp. 96 and 109.)

Mineral Matter.—The mineral matter of cow's milk varies according to different analyses, and is influenced to some extent by the food. The analysis of Söldner is compared in the following table with that of Bunge for human milk, showing the contrasts of the relative percentages. The figures are those quoted by Hammarsten.⁷

TABLE 39.—PERCENTAGES OF MINERAL MATTER OF COW'S MILK AND OF HUMAN MILK

	Cow's milk, Söldner	Human milk, Bunge
Potassium oxide.....	0.172	0.0703
Sodium oxide.....	0.051	0.0257
Calcium oxide.....	0.198	0.0343
Magnesium oxide.....	0.02	0.0065
Ferric oxide.....	0.00035 ⁸	0.0006
Phosphoric oxide.....	0.182	0.0469
Chlorine.....	0.098	0.0445

The mineral matter of cow's milk differs from that of human milk chiefly in the much larger total amount present, in the greater proportion of phosphorus and of lime, potash, and magnesia, and the smaller quantity of iron. Part of the lime is combined with the casein; the rest is united with phosphoric acid to form phosphates which hold the casein in suspension in the milk.

Ferments, Extractive Matter, Etc.—Cow's milk differs further from human milk in the presence in it of a much larger percentage of citric acid (cow's milk 0.2 per cent.; human milk 0.05) and a much smaller amount

¹ Arch. f. d. gesammte Physiol., 1894, LVI, 577.

² Dairy Chemistry, 1899, 120.

³ Zeitsch. f. phys. Chem., 1896, XXII, 211.

⁴ Loc. cit., 153.

⁵ Journ. Amer. Chem. Soc., 1893, 605.

⁶ Phila. Med. Journ., 1901, Feb. 2.

⁷ Phys. Chem., 1904, 455; 460.

⁸ Bunge, Zeit. f. Biol., 1874, X, 309.

of lecithin and of nucleone. A number of ferments characteristic of human milk are, however, absent from that of the cow. (See p. 98.)

Bacteria in Cow's Milk.—All cow's milk, as ordinarily supplied, contains numerous bacteria. Even when first drawn from the udder this is the case, although it is true that by far the greatest number of bacteria are found in the first part of the milk, the latter portion being nearly free. In addition to the germs within the udder, direct contamination of the milk occurs through the dust of the stable, dust from the cow's udder and belly, contaminating cow-manure, the hands of the milker and the like; and, inasmuch as milk forms one of the best culture media, the multiplication of these is extremely rapid and the number of varieties great. Park and Holt¹ isolated and studied 239 species. The average market-milk contains from 2,000,000 to even 10,000,000 or more bacteria to the c.c. (Bergey);² a number often greater than is found in the sewage of our large cities (Rosenau).³ The milk produced in dairies managed with sufficient care need never contain more than 10,000 to the c.c., while an average of from 2000 to 2500 is perfectly possible. The varieties which may be present are very numerous, depending upon the surrounding sources of contamination. According to Bergey those derived from the udder are forms of staphylococcus and streptococcus and the pseudodiphtheria bacillus. Those entering in other ways may be classified into *non-pathogenic* and *pathogenic*.

Of the *non-pathogenic* the most frequent are the lactic acid-producing bacteria, developing lactic acid by acting upon the sugar. Prominent among these are the streptococcus lacticus, the bacillus lactis acidi and, less frequently, the bacillus lactis aërogenes. These germs enter the milk during milking or afterward, at the same time with others, some of which are often displaced by the production of an acidity which is unsuited to their growth. A second class is the butyric acid group, breaking up the sugar and fat and producing butyric acid. A third group is composed of the proteolytic bacteria which, usually after coagulating the milk, may cause a breaking up of the protein. Many of these are also capable of producing lactic acid, as for instance the bacillus coli, which is the one of this group most commonly present. The bacillus proteus, bacillus alkaligenes and certain other germs belong to the proteolytic class. Numerous other organisms may be present, among them pyogenic germs derived from a diseased udder, bacteria giving rise to various discolorations of the milk, or others which give it a bitter or other unpleasant taste. To a certain extent the lactic-acid germs may not be harmful if in small amount, since the acid produced serves to check the growth within the intestine of the putrefactive bacteria.

A large variety of *pathogenic* germs has been found at times in milk, and epidemics have repeatedly been traced to this source. Kober⁴ collected 330 outbreaks of infectious diseases traceable to the milk-supply. Infection through milk is true especially of typhoid fever, scarlet fever and diphtheria. Epidemics of septic sore throat have been produced in the same way. Tubercle bacilli of the bovine type may also be found in cow's milk if the udder is tuberculous, although they occasionally occur even when it is healthy, in this case the germs entering the milk after milking. The human type of tubercle bacillus is probably the more

¹ Med. News, 1903, LXXXIII, 1066.

² Univ. of Penna. Med. Bull., 1904, July-August.

³ Hygienic Lab. Bull., 1912, No. 56, 429.

⁴ Amer. Jour. Med. Sci., 1901, May.

frequent in cow's milk, entering it from the dust of the air, the hands of a tuberculous milker, or in other ways. Among other pathogenic germs occasionally occurring in milk, and in some instances producing disease in infants and children, are those of cholera, dysentery, and anthrax.

The number of bacteria in cream is greater than in milk, even when this is obtained by centrifugating and with all precautions against contamination. Nevertheless the experience of the Milk-Commission of the Philadelphia Pediatric Society¹ has demonstrated that a standard of 25,000 germs to the c.c. of centrifugated cream is not too rigorous. When cream is obtained by the gravity process the number of bacteria is decidedly increased. In comparing the different layers of top milk, the greatest number of bacteria are found near the top.

By the heating of the milk many of the germs can be destroyed. Many of the forms of lactic-acid-producing bacteria are destroyed at a temperature of 60°C. (140°F.) as are most of the non-spore-bearing pathogenic germs. The tubercle bacillus is resistant, but will yield to a temperature of 155°F. (68.3°C.) continued for 30 minutes. Rosenau² found it no longer infectious when heated to 60°C. (140°F.) for 2 minutes. The butyric-acid bacilli and the spore-bearing proteolytic germs require a higher temperature, and even that of boiling does not destroy the spores. Consequently it has been believed that the temperatures usually employed for pasteurizing milk, which destroy the lactic acid bacteria, give a fuller opportunity for the subsequent growth of the dangerous proteolytic germs, because there was no later development of acidity to check their growth, and no warning of danger by the milk turning sour; and that especial care was necessary to prevent such a growth in milk which had been heated in this way. It has been shown, however, by Ayers and Johnson³ that not all of the germs producing lactic acid are killed at a temperature less than 168°F. (75.5°C.), and that consequently there is no special opportunity given for the development of the proteolytic bacteria. In fact, the majority of both the acid-producing and the proteolytic germs are destroyed at this temperature, and the subsequent development of both sorts occurs in heated milk with the same degree and with the same relationship as in raw milk. It has been recommended by Budde⁴ to add small amounts of hydrogen peroxide to the milk, and then to heat this to from 50 to 52°C. (122 to 125.6°F.), the object being to destroy the bacteria more completely. It is not certain that the process is as entirely harmless as claimed.

Pus in Milk.—All cow's milk shows under the microscope a certain number of leucocytes. As a result of the study of a large number of cows, Bergey⁵ and Stokes⁶ concluded that the presence of more than 10 leucocytes per field of a $\frac{1}{12}$ emersion lens, obtained by centrifuging 10 c.c. (0.338 fl.oz.) of milk, constitutes pus, especially when the cells are grouped in small masses and accompanied by chains of streptococci. There is, however, a very great range in the number of cells found in milk from healthy cattle, and the determination of the presence of pus by numbers alone is unsatisfactory (Lewis).⁷

¹ Arch. of Ped., 1904, XXI, April.

² Hyg. Lab. Bull., 1912, No. 56, 684.

³ Bull. 126, Dept. of Agriculture, Bureau of Animal Industry.

⁴ Milchzeitung, 1903, No. 44, 690. Ref. Kastle and Roberts, Hygienic Lab. Bull., 1912, No. 56, 385.

⁵ Dept. of Agric. of Penna., Bulletin No. 125.

⁶ Ann. Rep. Health Dept. of Baltimore, 1898. Ref. Bergey.

⁷ Amer. Jour. Dis. Child., 1913, VI, 225.

Cream.—Cream is to all intents merely a super-fatted milk, and, strictly speaking, any milk containing more than 4 per cent. of fat should be called cream. It is true that as the percentages of fat increase, those of sugar and of mineral matter diminish slightly. Regarding the protein there has been some difference of opinion. Wentworth¹ maintained that the amount of protein is greater in cream than in milk. Adriance,² on the other hand, and Richmond³ claim that the percentage decreases as that of fat increases, and this is the generally accepted view. There is considerable discrepancy in the statements made regarding the degree of diminution in the percentages of protein and sugar with increasing strength of the cream in fat. The following table represents approximate averages based upon the figures given by a number of investigators:

TABLE 40.—PERCENTAGES OF FAT, SUGAR AND PROTEIN IN CREAMS OF DIFFERENT STRENGTHS

Per cent. cream	Fat	Carbohydrate	Protein
32	32	3.40	2.5
20	20	3.90	2.9
16	16	4.20	3.05
12	12	4.30	3.20
10	10	4.40	3.30
7	7	4.45	3.40

In the proportions to which the cream ordinarily employed must be diluted to obtain a sufficiently small amount of fat, the variation of the percentages of the other solids is immaterial and may be disregarded. Cream is obtained either by skimming or dipping, in which case it is called "gravity cream," or by the use of the "separator" and then called "centrifugated" or "separator" cream. Practically all the cream sold on the market is separator cream. By this process it may be made of almost any strength desired up to 30 per cent. or even 40 per cent. or more of fat, by regulating the speed of the machine. The centrifugated cream has the great advantage that it can be prepared almost as soon as the milk is obtained from the cow and is consequently much freer from bacterial growth. A disadvantage claimed is that the centrifugal force breaks up the emulsion and is harmful to the infant. I have never witnessed any injury from this source, and the careful experiments of White and Ladd⁴ indicate that no difference exists between mixtures made from gravity cream and those from centrifugated cream, and that the separation of visible globules of fat sometimes seen in bottles prepared for infant-feeding may occur with either and is due to other causes, especially the combined influence of heat and the shaking during transportation.

Top-milk.—This is the term applied to any number of the upper ounces of the milk in a milk-jar after having stood a number of hours. Strictly speaking top-milk is cream, of a strength varying with the number of ounces of milk which is removed from the jar. As, however, the lumpy persist in regarding as cream only the part which has separated with a distinct line of demarcation from the milk below, and as not infrequently it is desirable to remove more than this, the employment of the title "top-milk" is preferable. It designates the amount of milk removed,

¹ Boston Med. and Surg. Journ., 1902, CXLVI, 683; 1903, CXLVII, 5.

² Arch. of Ped., 1900, May; 1904, Jan.

³ Dairy Chemistry, 1899, 215.

⁴ Phila. Med. Journ., 1901, Feb. 2.

whether only 1 oz. or 31 oz. from the quart-jar of milk. Even with the precaution in the use of terms, and in spite of directions which seem explicit, mothers frequently persist in removing from the jar only the number of ounces which they require instead of the number ordered by the physician. It is manifest that the strength of the top-milk in fat is in inverse proportion to the number of ounces removed, and that a very decided difference in the milk-mixture results if, for instance, the top 2 oz. are taken instead of 2 oz. from the top 8 or more of the quart after removing all of this.

To obtain top-milk, a quart of milk, as soon after milking as possible, is strained into one of the ordinary quart milk-bottles, closed, cooled, and then kept on ice or in ice-water for 6 hours or longer. In dairy milk delivered in jars the cream has already risen, and there is no need for the



FIG. 20.
—CHAPIN
DIPPER.

bottles to stand longer before removing the top-milk, if the cream-layer is sharply defined. To obtain the top-milk one may employ syphoning, pouring, or dipping. With the former a glass tube bent into the form of a syphon is filled with sterile water, the long end of this is kept closed with the finger, and the short end then inserted carefully into the jar until it reaches the bottom. The finger is now removed, the water allowed to flow out and the milk which follows received in a graduated vessel, the "top milk" meanwhile sinking slowly toward the bottom of the jar. When the desired number of ounces remain in the jar, as determined by observing how many have escaped, the syphon is removed. Great care must always be taken that the jar, syphon, and other vessels are scrupulously clean. Sucking the water through the syphon by the mouth, in order to start the flow is, of course, out of the question. A much better and easier method is the employment of the dipper devised by Chapin¹ and holding 1 oz. of milk² (Fig. 20). In employing it enough of the top-milk is first skimmed off with a teaspoon and placed in the dipper to allow using the instrument without causing the milk to overflow. As many ounces are then dipped off as are required. By letting down the dipper carefully and slowly into the milk the top layer is disturbed practically not at all. A modification of the dipper has a displaceable bottom which allows of the instrument being pushed into the jar without danger of causing an overflow. The original device is, however, preferable in many respects.

Still another method consists in pouring off carefully the number of ounces of top-milk desired. This has been shown by Townsend³ to give a top-milk with a percentage somewhat less than that obtained by the other methods. As it involves a different calculation it is not to be recommended.

The great advantage of top-milk is its economy and its convenience when separator cream of known strength in fat cannot be obtained. Its disadvantages are that it contains a greater number of bacteria, and that the fat-percentages in it are subject to decided variation, investigators differing somewhat in opinion regarding this. The strength undoubtedly

¹ New York Med. Journ., 1899, Nov. 4.

² This dipper can be purchased through druggists, or from the Cerec Co., Tappan, N. Y.

³ Boston Med. and Surg. Journ., 1903, CXLVIII, 412.

varies with different milks, those rich in fat, such as Jersey, giving a higher fat-percentage in a definite number of ounces of top-milk. Due allowance must be made for this. Thus, if a milk containing 5 per cent. of fat is employed, the removal of the upper 2 oz. of the quart after standing reduces the fat to 4 per cent. Further removal by dipping may now be done, taking the number of ounces which would have been used had the milk originally been of 4 per cent. fat-strength. That is to say, the "upper 8 oz.," for instance, would mean the upper 8 of the 30 oz. remaining in the jar after the top 2 oz. had been removed. The results of a series of unpublished examinations of the fat-strength of top-milk made by Dr. Chas. A. Fife, combined with a number of estimations published by others have led me to the adoption of the top-milk strengths which will be referred to later under Calculation of Milk Formulæ. (See p. 139.) These give results sufficiently accurate for practical purposes.

Skimmed Milk, Bottom Milk, Fat-free Milk.—The milk remaining after any of the cream has been removed by skimming or by dipping off the cream-layer is denominated skimmed milk or bottom milk. Its percentages of sugar and of protein are slightly higher than, but for practical purposes the same as, those of whole milk. If 2 oz. of the top is removed, the fat-strength equals 3 per cent.; if all the milk below the cream-layer is used the fat-strength is about 1 per cent. If all but the lower quarter of the jar is taken away the percentage is less, frequently then being from 0.2 to 0.5 per cent., and the milk may be considered as fat-free. (See p. 139.) Actual fat-free milk, however, can be obtained only by the use of the separator. A comparison of the relative strengths of the different elements in whole milk, skimmed milk and fat-free milk, as given by Morse and Talbot,¹ may be seen in the following table:

TABLE 41.—COMPARISON OF WHOLE MILK, SKIMMED MILK AND FAT-FREE MILK

	Fat	Sugar	Protein
Whole milk.....	4.0	4.5	3.50
Skimmed milk.....	1.0	5.0	3.55
Fat-free milk.....	0.25	5.0	3.65

Requirements in Good Milk.—There are certain requisites which must be insisted upon in order to make cow's milk a safe and satisfactory food. First of all it must be clean. The centrifugating process applied to ordinary cow's milk obtained without sufficient care shows that this is far from being true of it. Cleanliness with regard to contamination by bacteria is important. There should be an absence of pathogenic germs and but a small number of those of other sorts. The milk should be tampered with in no way, as by the addition of such preservatives as boric acid and formaldehyde, the removal of any of the cream, or the addition of water. Much of that furnished is very decidedly over 24 hours old before it is used. If it has been pasteurized it should be distinctly stated on the bottle that this has been done. An unusually low bacterial count in milk not known to have been produced with especial care suggests the employment either of chemical preservatives or of heat. The milk should be kept constantly at a temperature not over 45 F. (7.2°C.) from the time it is bottled until it is prepared for use, and the

¹ Diseases of Nutrition and Infant Feeding, 1915, 217.

time of transportation should be as brief as possible. Uniformity in strength is necessary, with conformity to the analytical standards of normal milk. If the cow's milk varies materially it is manifestly impossible to modify it in a way which will agree with the child to whom it is fed. If it is from cows which are ill its quality becomes impaired, or it may become decidedly harmful to the infant. Consequently mixed herd-milk is to be preferred to that from a single cow, as less liable to exhibit sudden alterations in strength.

Dairy Methods.—The best methods of dairy management must be followed to ensure the fulfilling of these requirements for good milk. The cows should receive a carefully adjusted diet and be exercised daily. They should not be allowed to pasture at large, eating what they may find. They should not be fed immediately before milking on account of the dust produced thereby. The milk should be from a mixed herd, preferably of "grade" cattle. Jersey cattle produce a milk too rich in fat; Holstein and Ayresshire one rather poor. The stable must be well ventilated, commodious, dry, screened, and kept strictly clean, and the cows so stalled that they cannot soil themselves or reach their udders with their heads. They should be cleaned and brushed daily, but not just before milking, and the hair upon the bellies, udders, inside of the thighs, and tails should be clipped short. The udders and teats should be washed with sterilized water before milking, and the milk-men should dress in washable sterilized clothing and caps, and wash the hands before the milking of each cow. Vessels of any kind into which milk enters must be kept bacteriologically clean. The milk-pails should be of special design intended to prevent the ready entrance of dust from the cow's belly or from the air of the room. Immediately after milking the milk should be rapidly cooled, strained; put in sterilized milk-jars and sealed. The cows should be in good health, and, to insure this, frequent veterinary examinations must be made, both of the animals and of their surroundings, and all those affected by any disease, especially by a suppurating affection of the udder or by tuberculosis, must be promptly withdrawn. Jersey cattle are claimed to be particularly prone to tuberculosis in this country, and all highly bred cows are, as a rule, of less vigorous health than "grade" cattle. Strict precautions, too, must be taken against the existence of any infectious disease among the employees of the dairy or their families, lest such become communicated to infants through the milk.

Certified Milk.—The evident lack in ordinary dairies of the fulfillment of any such regulations as those just mentioned, led to the establishment in various cities of Milk-Commissions, which in 1907 associated themselves as the American Association of Medical Milk-Commissions. The requirements detailed above constitute in most respects a partial abstract of the regulations adopted by the association. The purpose of a Milk-commission is to prescribe rules and regulations for dairies, with which a contract is to be made, and to see by inspection that the prescribed requirements and agreements are fulfilled. Personal systematic inspection of the dairy is made from time to time by members of the Commission, and repeated examinations of the buildings and the cows conducted by the Commission's veterinarian, and of the milk by its chemists and bacteriologists. To dairies the product of which fulfills the requirements a certificate is given to that effect, and such milk is properly called "certified milk." It is hardly necessary to say that much milk is advertised and sold by dealers as "certified" which in no way belongs in this

category, the certifying being either a fiction or done by those employed by the dairy for this purpose.

Properly certified milk usually and rightly sells at an increased price, since the cost of its production is necessarily increased. With sufficient ordinary care, however, a great improvement in the quality of ordinary market-milk can be made without any material increase in the expense.

GENERAL PRINCIPLES OF SUBSTITUTE FEEDING

Comparison of Human and Bovine Milk.—Before proceeding to the study of the preparation of milk-mixtures, we may conveniently place before us in tabular form the contrasts between human and bovine milk which have already been considered. The figures are approximate ones:

TABLE 42.—PRINCIPAL CONTRASTS OF AVERAGE BOVINE AND HUMAN MILK

	Cow's milk	Human milk
Specific gravity.....	1032	1030-31
Color.....	White.	Bluish-white.
Bacteria.....	Always present.	Practically none.
Coagulation by rennet and acid	Readily and produces firm curds.	Less easy and produces soft, flocculent precipitate.
Reaction to litmus.....	Generally acid.	Alkaline or amphoterie.
Fat.....	4 per cent.; containing more volatile fatty acids.	3.5-4 per cent.; containing less volatile and more oleic acid.
Sugar.....	4.5 per cent.	7 per cent.
Total Protein.....	3.5-4 per cent.	1-1.50 per cent.
Total Lactalbumin.....	0.3 per cent. ¹	0.5 per cent. ¹
Total Casein.....	3.0 per cent. ¹	1.2 per cent. ¹
Mineral matter.....	0.7 per cent.	0.2 per cent.
Total Solids.....	12-14 per cent.	12-13 per cent.
Water.....	86-88 per cent.	87-88 per cent.

The figures for lactalbumin and casein are those of Lehmann.¹ Reference to pp. 96 and 112 shows the variation among investigators as to the relative proportions of these two elements. All indicate, however, the relative increase in the proportion of lactalbumin in human milk as compared with cow's milk.

Modification of Milk.—The object in adapting cow's milk to infant-feeding is to change or *modify* it in some way, since without this change it is not tolerated by the majority of infants in the 1st year of life. A widespread misconception exists among the laity regarding this. By them "modified milk" is often spoken of as though it were some specific sort of food. As a matter of fact any element taken from or added to milk modifies it, even the mere addition of water. Modification is, therefore, a wide term indicating any change produced artificially in the milk. It becomes obvious from the study of the tabular composition that cow's milk in its natural state is far from suited to the healthy infant during the 1st year of life. Simple dilutions alter the proportions of all the solids uniformly, and are sufficient in some instances, while in others other methods are required. In brief the objects of modification are chiefly to prevent the development of bacteria or to reduce the number already present; to prevent the formation of the firm, large rennin curd; to correct the acid reaction; to reduce the protein, and sometimes to dimin-

¹ Lehmann, Arch. f. d. gesammte Physiol., 1894, LV1, 577.

ish especially the casein by the employment of whey; to reduce the salts; and to maintain or lessen the percentage of fat, this reduction being frequently necessary on account of the large proportion of volatile fatty acids in cow's milk. Theoretically the accomplishment of such changes seems easy. Practically it is impossible, no matter what we do, to produce a perfect imitation of human milk. This should be recognized at the outset. The differences in the character of the fat cannot be satisfactorily altered by any modification; the biological characters of the casein are not the same; the ferments are different. So, too, although it would appear at first sight that the nearer the percentage of the food-elements corresponded with those of human milk, the better suited the food would be for the infant, experience has taught that this is far from the truth; owing in part to the different ultimate constitution of the various elements, and in part to the individuality of the infant. No one formula can be expected to suit even the healthy child; and the problem of adapting the requirements to the infant with digestive disturbance is many times greater.

Percentage-feeding.—With the growth of a fuller knowledge of the composition of cow's milk came the expansion of the principles of what has been called "Percentage-feeding." The percentage-method is not in itself new. Biedert¹ emphasized its importance in a general way; and still earlier, in 1858, Cummings² gave a clear exposition of it. There had been, however, no special stress laid upon the importance of modifying as required the percentages of one or another ingredient of the milk-mixture, and no method devised for a ready accomplishing of these alterations, until the writings of Rotch³ brought the matter into prominence. Percentage-feeding marked a great advance in the scientific feeding of infants. Physicians had for years prescribed modified cow's milk; *i.e.*, they had ordered a varying number of ounces of milk diluted with water, adding, it may be, cream and sugar, and changing these modifications to suit the case. The results, of course, were percentage modifications of the cow's milk ingredients, yet what these were the prescriber usually did not know. The method was inexact because the *thought* was in terms of such composite articles as "milk" and "cream," and to obtain accuracy in composition and in results was impossible in this way. In the percentage method one thinks in definite percentages of "fat," "sugar," "protein," etc., determining how much of each the individual infant requires. The percentages thus chosen are then transformed into terms of the number of ounces of milk, water, cream, etc., required to be equivalent to these. (See p. 137 *et seq.*). The great advantages are increased accuracy of thought and the consequent better results to be expected. The problem, for instance, of how to increase the protein of a milk-mixture without increasing the fat can be solved only by thinking in percentages.

The percentage method has suffered from much lack of comprehension and from misstatement. It has been looked upon as an innovation, considered complicated, and called by European writers the "American method." There is, in fact, nothing complicated about it, and any school-boy who has studied the rules of proportion, possesses all the mathematical knowledge required. As to its newness, it is, as stated, not new. The

¹ Untersuch. ü. d. chem. Unterschieden d. Menschl- u. Kuhmilch, Inaug. Dissert., Giessen, 1869.

² Amer. Jour. Med. Sci., 1858, XXXVI, 25. Food for Babies, 1859.

³ Brit. Med. Journ., 1902, Sept. 6.

only thing really new in it was the *method of thinking*, by which the food-mixture is built up synthetically from its elements to attain the desired chemical composition. It has nothing whatever to do with the choice of the food-elements, and is in no sense a *method of feeding*. It merely records with approximate accuracy the percentage amount of these elements employed, and enables a change readily to be made in them according to the requirements of the case. Much, too, has been written in recent years regarding the superiority of the so-called "simple dilution" of milk over percentage-feeding. This shows a total lack of comprehension of the subject. There can be no possible contrast or contradiction between them. Any physician is entitled to use whole milk diluted with water, and with the addition of sugar, in any amount the case requires, and in fact such dilutions are all that is needed in many instances. The important matter for scientific feeding is to realize the percentages of fat, sugar and protein which are obtained in this way; and such recognition makes the method a percentage one; and in any event the proper amount of sugar to be added has to be determined by a percentage calculation. The fact is not to be forgotten, however, that the normal healthy baby needs and can digest fat; and that the invariable employment of diluted whole milk reduces this element below the infant's capacity and requirements, and forces the use of other elements in excess to make up the caloric deficiency. Without the knowledge of percentage composition no scientific infant-feeding is possible.

It is to be understood that the percentage method is not supposed to give absolutely accurate statements of the amount of the various food-elements present. This is not, however, a matter of practical importance. It is not so much minute knowledge of the *initial composition* which is desired, as the ability to increase or decrease with comparative accuracy one or more of the ingredients at will, according to the necessities of the case and guided by the knowledge of the mutual relationships which the elements bear to each other. It is evident, too, that the employment of the "caloric method," to be described in the next section, is, in the nature of things, an impossibility without the knowledge of the percentage composition of the milk-mixture.

The "Caloric Method" (See also p. 52).—This in its origin was simple. Heubner¹ as a result of careful experimental work upon healthy breast-fed infants, came to the conclusion that 100 calories per kilogram of body-weight of the breast-fed child should be rendered daily by the food in the first part of the 1st year, or 120 calories in the case of artificially fed children, with a gradual progressive diminution in number during the year. This figure expressive of the division of the total caloric need by the weight of the infant he called the "Energy-quotient." In *avordupois* it is expressed by 45 calories per pound of body-weight. Heubner then adopted a series of different dilutions, using $\frac{1}{3}$, $\frac{1}{2}$, or $\frac{2}{3}$ of whole milk in water, and merely added a sufficient percentage of sugar and starchy material to make up the calories to the required normal amount. Various later investigators have confirmed his views, although with considerable difference of opinion as to the figures which most accurately express the energy-quotient; and various other calculations have been made permitting the use of other than these simple dilutions, yet based upon a calculation of the calories contained. The energy-quotient at present very commonly adopted for breast-fed and artificially fed infants is 100 for the first quarter of the 1st year, 90 for the second, 80 for the third,

¹ Zeit. f. diät. u. physikal. Therapie, 1901, V, 13.

and 70 for the fourth quarter. In avoirdupois weights these would be approximately equivalent to 45, 40, 35 and 30 calories, respectively, per pound of body-weight.

The method is of great advantage in that it enables one promptly to determine whether or not a normal infant may be considered to be under-fed or over-fed as far as calories go. It has, however, distinct disadvantages, if followed too implicitly. Not only is there, as stated, a discrepancy in the definition of what actually constitutes the energy-quotient for normal infants, but for premature and emaciated infants the figure given by Heubner may be distinctly too low, inasmuch as it is based upon the *weight* of the child and not upon its *body-surface* or *body-bulk* as it should be. On the other hand, the activity of the child is to be considered; one exhibiting free movements of the body, or crying much, having a greatly increased heat-dissipation, and requiring, consequently, more calories; while a marantic infant whose movements are sluggish, feeble and infrequent, and whose life-forces are at a low ebb may be sufficiently nourished by food containing distinctly fewer calories than would have been expected. The difference, too, between the sleeping and the waking state is decided. We have no means of knowing, therefore, what the energy-quotient really is for infants other than those in health, and it is probable that the requirements vary with the child (Howland.)¹ The greatest disadvantage of the method, if followed strictly and not as its originator intended, is the tendency for the physician to take too little account of the relative assimilability of the different food-elements, but to regard it as a matter of indifference which is employed, so long as the food contains sufficient calories. Of course in actual practice no experienced pediatricist would act according to this; but the less experienced physician may readily be led into the mistake. It is evident that a food may contain the requisite number of calories and yet be constructed upon an entirely wrong basis. Protein, for instance, might have been left out completely, and the caloric requirements still be met; but on such a food life would be impossible. On the other hand, the caloric requirements may be supplied by fat in a percentage-mixture of a fat-strength which would cause active indigestion in the healthiest baby. The method is, therefore, like that of percentages, not a *method of feeding*. It is only one of calculation, serviceable in estimating whether or not the food answers the infant's caloric needs. It aids in no way in determining how the food shall be composed in order that these needs shall be met.

If perfectly understood there can be no possible conflict between the two so-called "methods." The percentages of the elements of the food should first be selected according to the digestive capacity of the infant, and the caloric value of these then calculated to determine whether the energy-requirements are fulfilled.

With the accepted data that 1 gram of fat furnishes in the economy 9.3 calories; and 1 gram of protein and of carbohydrate each 4.1 calories, it is readily possible to estimate the total number of calories in a litre of any milk-mixture, if the percentage composition is known. Thus supposing an infant is receiving a litre of a food-mixture containing fat, 3 per cent.; sugar, 6 per cent. and protein, 1.5 per cent.: If we multiply 1000 grams by each of these figures we obtain as present in the food:

Fat	=	1000 × 0.03	=	30 grams
Sugar	=	1000 × 0.06	=	60 grams
Protein	=	1000 × 0.015	=	15 grams

¹ Amer. Journ. Dis. Child., 1914, V, 393.

If now we multiply each of these amounts by the number of calories which 1 gram produces, we obtain:

Fat	=	30×9.3	=	279.0 calories
Sugar	=	60×4.1	=	246.0 calories
Protein	=	15×4.1	=	61.5 calories
Total in 1 litre of food = 586.5 calories				

Since the infant requires in its food 100 calories per kilogram of its body-weight, we find that 586.5 calories divided by 100 = 5.865; *i.e.* the weight in kilograms of the infant which should thrive upon this quantity of food, so far as the caloric value is concerned.

When the infant is fed and weighed, as is customary in English speaking countries, in terms of ounces, a fourth calculation is required reducing the metric to *avoirdupois* system. As this is inconvenient, it is better to make the calculations from the beginning in English measures. A very simple formula has been calculated by Fraley¹ which gives results quite sufficiently accurate for practical purposes. This formula in brief is

$$(2F + S + P) \times 1\frac{1}{4}Q = C$$

or, in detail, twice the fat-percentage, plus the sugar-percentage, plus the protein-percentage, multiplied by one and a quarter times the total quantity of food-mixture given in the day, equals the total number of calories furnished by the day's food. Supposing, for example, a milk-mixture contains fat, 3 per cent.; sugar, 6 per cent.; protein, 2 per cent.; 6 bottles of 6 oz. each administered; *i.e.*, 36 oz. in the 24 hours. Substituting these figures the formula will read $(2 \times 3 + 6 + 2) \times (1\frac{1}{4} \times 36) = 14 \times 45 = 630$ calories. Dividing this by 45, the number of calories required per pound of body-weight, we have resulting 15 pounds. That is to say, the formula should be sufficient for a child weighing 15 pounds. A modification of the Fraley formula (Holt and Howland)² may be usefully employed in another way, to estimate the caloric value of each ounce of the food used. In the form of an equation it reads as follows: $(2F + S + P) \times 1.3 = \text{No. of calories in an ounce of the food.}$

A knowledge of the caloric value of milk and milk-derivatives, as well as of various other articles of diet suitable for infants and children, is often of great value. A list of such equivalents will be found later (p. 175).

Details for Milk-modification.—With the differences between cow's milk and human milk in view, as shown in the table already given (p. 119), we may take up the method to be employed to eliminate these as far as is necessary.

Altering the Reaction.—This is best done by the addition of lime-water. An amount equal to at least 5 per cent. of the total food, *i.e.*, $\frac{1}{2}$ oz. to each 10 oz., will be needed to make the alkalinity of a cow's milk-mixture equal approximately that of human milk. If for any reason lime-water is not employed, bicarbonate of soda may be substituted, 1 grain of this being equivalent in alkalinity to 1 oz. of lime-water. The action of lime-water or other alkali after its ingestion is not thoroughly understood, and it is questionable whether in the present state of our knowledge any alkali is necessary or advisable. The lime-water, it is true, neutralizes the acidity of the cow's milk, as also later the hydrochloric acid secreted in the stomach, and delays or prevents the

¹ Arch. of Ped., 1912, XXIX, 123.

² Diseases of Children, 1916, 181.

formation of large, tough protein curds (see p. 109); and it has been believed that in this way it favors the more rapid emptying of the stomach. To accomplish any inhibition of coagulation within the organ it must, however, be given in much larger amount than is ordinarily employed (25 to 50 per cent., Morse and Talbot),¹ and the increasing of alkalinity in this way would result, according to Cowie and Lyon,² in delaying the opening of the pylorus and the exit of the milk through it. Clark³ maintains that the addition of lime-water to the food in reality stimulates the stomach to an increased secretion of hydrochloric acid. It is evident, therefore, that our knowledge of the action of lime-water is far from complete. It is a mistaken idea that lime water should be added in order to supply mineral matter for the proper development of the osseous tissues. This is entirely unnecessary.

Preventing the Formation of Firm Casein Coagula.—This is sometimes necessary, and is to be accomplished in various ways. The use of alkalis, as stated, aids in the matter. Citrate of soda has been much employed to prevent the coagulation, giving generally about 1 grain for every ounce of milk. Boiling the milk is another efficacious method (p. 109); or the total amount of casein in the mixture may be reduced, and the deficiency of protein made good by the giving of lactalbumin in the form of whey (see p. 145). The addition of a cereal diluent instead of water mechanically prevents the formation of tough coagula, acting, it is believed, as a protective colloid. From 0.60 to 1.20 per cent. of starch should be employed. (See p. 152.) Peptonizing the milk may be used in some cases (p. 146), or buttermilk given (p. 147), since the casein in this is not affected by rennet. In casein-milk also (p. 148) the casein is in a form in which large, tough curds cannot develop.

Altering the Proportions of the Milk-elements.—Should it be the intention to alter the composition of cow's milk to make it more closely resemble human milk—a procedure not always to be desired—the reduction of the percentage of the proteins is a simple matter if we treat these bodies as a unit. It is accomplished by merely adding to the milk some attenuant, generally water, in the proportion of 2 or 3 times its volume. If, however, we have regard to the composite nature of the protein-element, and wish to approach the composition of human milk more closely than simple dilution accomplishes, it is necessary to reduce the percentage of casein while leaving the lactalbumin unaffected. This can be done by the use of whey-mixtures, since the casein has been removed from them by the action of rennin. The employment of whey for this purpose will be described later (p. 145).

Disregarding the difference in the proteins, and with the intention of simulating human milk in other respects—a procedure, as stated, not always to be desired—it will be noticed in consulting the table on p. 119 that in diluting cow's milk with 2 or 3 parts of water the protein-percentage sought for is reached, and that the salts likewise are reduced sufficiently for all practical purposes. Simultaneously, however, the amount of fat will be made decidedly below the percentage present in human milk, and that of sugar still more so. The obvious remedy is to add sugar and fat to the mixture. The percentage of sugar is increased merely by adding enough to bring the figure up to the required amount. The percentage of fat may be maintained either by diluting a top-milk

¹ Diseases of Nutrition and Infant Feeding, 1915, 204.

² Amer. Jour. Dis. Child., 1911, II, 252.

³ Amer. Jour. Med. Sci., 1909, CXXXVII, 872.

of such an initial strength that when the protein is sufficiently reduced the fat will still be of a higher percentage than it; or by replacing a certain amount of the whole milk used in the mixture by an equal quantity of a strong cream of known strength. The simple calculation necessary to reduce the percentage of protein, maintain that of the fat and increase that of the sugar, will be more fully discussed in considering Home Modification. (See p. 137.)

Removal of Bacteria. Sterilizing. Pasteurizing.—Much better than the removing of bacteria is the prevention of their entrance. In the best milk, obtained by the method previously outlined (p. 118), often no treatment of any sort is necessary, the germs being so few in number that the infant can tolerate them well. With ordinary milk, however, and even with the best milk in the hottest summer weather, some procedure is needed. It must be remembered that no degree of heat can do more than partially destroy the harmful toxins which may have been formed already, and which are the cause of many illnesses. Milk which contains these cannot be made fit to use.

Sterilization.—The term *sterilization* as commonly applied to milk consists in the subjection of it for a short time, $\frac{1}{2}$ to 1 hour, to a temperature of 212°F. (100°C.). This is not strictly a sterilization, since some varieties of bacteria remain, and spores of others are not destroyed at this temperature. The first practical "sterilizer" for household use in the feeding of infants was that devised by Soxhlet.¹ Since then many have been invented, that of Arnold being very well known, all acting on the same principle; viz. the subjecting of the milk in closed bottles to the action of steam, thus raising the food to a temperature of nearly or quite 212°F. (100°C.). The bottles, previously boiled, are filled with as much of the milk-mixture as is needed for each feeding; the necks carefully wiped and then stopped with sterilized, non-absorbent cotton, and the filled bottles stood in the rack intended to hold them and this then placed in the receptacle and subjected to the action of steam for an hour. After this the bottles should be cooled as rapidly as possible, and when cool enough not to break put upon ice and kept there.

The effect of heat upon milk has already been described (p. 109). Sterilizing at the temperature of 212°F. (100°C.) causes a certain degree of decomposition of the sugar; precipitates the lactalbumin; produces some alteration of the casein, and interferes with its coagulation by rennin; probably alters to some extent the mineral matter; destroys some of the ferments, and decidedly lessens the bactericidal power, which would have tended to prevent the growth of bacteria for a time. There is reason to believe, also, that the use of sterilized milk may sometimes give rise to the development of infantile scurvy. Yet in spite of this list of apparently important alterations, the danger from sterilization is certainly over-rated. There is no positive proof that sterilized milk is, or is not, less well digested than is raw milk, in spite of the large amount of evidence offered upon each side of the question. It is certainly better to raise the milk to the temperature of boiling than to employ it raw if not of the highest quality.

Pasteurization.—This term had its origin in the employment by Pasteur of a temperature less than 100°C. (212°F.) for the preservation of wine. As applied to infant-feeding it consists in the subjection of the bottles of milk-mixture, prepared as for sterilizing, to a temperature of less than boiling; one of from 140 to 165°F. (60° to 73.9°C.) being ordi-

¹ Münch. med. Woch., 1886, XXXIII, 276.

narily recommended and the heat maintained for from 30 to 60 minutes. This is sufficient to kill the pathogenic bacteria and the larger number of the non-pathogenic organisms. (See p. 114.) It will not, however, destroy the spores of the proteolytic bacteria. The effect upon the chemical composition of the milk is certainly less than is the heating to the boiling point, and at 140° or even at 150°F. (60° to 65.5°C.) is practically absent; but mechanically a difference is produced, shown especially in the imperfect rising of the cream (p. 109). Particular care must be taken to cool the milk rapidly afterward and to keep it cold, otherwise the protein is decomposed by the bacteria which may develop, and the milk, although it may not sour, is rendered especially dangerous (Bergey).¹ Pasteurization is a delicate process, since a temperature higher than 150°F. (65.5°C.) possesses some of the disadvantages of sterilizing, while one lower than 140°F. (60°C.) does not act sufficiently upon the germs. To be efficient it must be done accurately. All such makeshifts as putting the bottles into a dishpan of hot water, heating in a sterilizer for a certain number of minutes, and the like, are to be condemned, unless a careful observation is made of the actual degree of temperature attained, and the duration of this. A sterilizer may, it is true, be made use of, taking care not to raise the temperature to that of boiling.

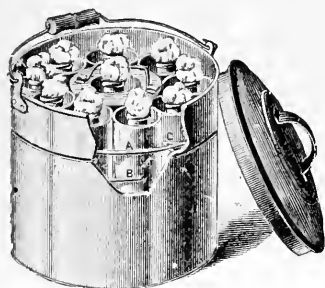


FIG. 21.—FREEMAN PASTEURIZER.

In this case, however, a thermometer must be employed and the temperature of the water constantly watched. This is a troublesome and time-consuming method and requires more knowledge than can be expected of an average nurse-maid and of many mothers. Various pasteurizers have been constructed. A very convenient and accurate one, easy to use, has been devised by Freeman (Fig. 21).² This consists of a metal pail with a groove about its circumference indicating the amount of water to be put into it, and a rack to hold the bottles, each bottle fitting into its own cylindrical metal cup, which, when water is poured around each bottle, prevents these from cracking when exposed to the heat. In the employment of the pasteurizer the pail is filled with water up to the grooved line, placed upon the stove or other source of heat, and the water raised to the boiling point, replacing any water which may have boiled away. Meanwhile the bottles are prepared as for sterilization and placed in the rack, and enough water poured into each cup to surround them. The milk in the bottles should be cold, and any bottles not needed for food should be filled with cold water and put in the rack. This is necessary to maintain the proper balance. The pail is now removed from the fire, set upon a wooden or other non-conducting table out of draughts, the rack put in, the lid put on, and the apparatus allowed to stand for 1 hour. In 10 minutes the temperature of the milk has risen to 55°C. (131°F.) and in 20 minutes from the beginning to slightly over 60°C. (140°F.), and this temperature is maintained for the remaining 40 minutes. The lid is now removed and cold water from the faucet is allowed to flow freely into the pail, overflowing into the sink. This produces the rapid cooling of the bottles which is neces-

¹ Proc. Path. Soc. Phila., 1905, 102.

² Med. Rec., 1892, XLII, 8; Arch. of Ped., 1896, 595; Jour. Amer. Med. Assoc., 1907, XLIX, 1740.

sary. When sufficiently cool they are at once put upon ice and kept there. The Freeman pasteurizer has the great recommendation that its employment is perfectly simple, and that it requires no watching whatever. Owing to the almost complete harmlessness of pasteurization, it should be employed with all milk not produced under the most careful supervision, and even with this it is advisable in hot weather.

What has been written above applies entirely to *home pasteurization*, which is the method always greatly to be preferred. In recent years *commercial pasteurization* has become very extensively used. Several procedures have been employed. The "flash" method passes the milk over hot pipes, heating it for a few seconds to the temperature of about 170°F. (76.7°C.). It is entirely untrustworthy. The "holding" method maintains the milk at 140 to 155°F. (60° to 68.3°C.) for 20 minutes or longer. This method is satisfactory if properly done; but in its commercial application it easily fails of attaining the desired temperature. As shown by Schorer and Rosenau,¹ the safest commercial method is to pasteurize in sealed bottles, but at least 30 minutes should be allowed to raise the milk to 145°F. (62.8°C.), and this temperature maintained for 30 minutes more. Certainly any pasteurizing done commercially without strict supervision is liable to be dangerous. The dealer is tempted to employ the cheapest method which will retard the growth of the lactic-acid-producing organisms and prevent souring; while for the physician souring is the least of the evils which may occur, and may be a valuable index that the milk is not fit for use. The proteolytic germs are far more dangerous to the infant. Further, the purchasing of pasteurized milk by the mother, independently of medical advice, gives her a false sense of security and engenders lack of sufficient care of the milk after it has been delivered. As a matter of fact, all pasteurized milk requires the same care in keeping it cold that applies to the handling of raw milk. Perhaps the greatest disadvantage of commercially pasteurized milk delivered in the bottle as whole milk, is the unsatisfactory manner, already referred to, in which the cream rises. This makes the employment of either top milk or skimmed milk subject to great uncertainty. The fat in the former is less than it should be, and that in the latter often so great that in cases of disturbed digestion a sufficiently low fat-percentage cannot be obtained.

The Action of the Different Food-elements.—Although a cow's milk-mixture which contains percentages similar to those of human milk, and which possesses the requisite number of calories, theoretically should agree with and properly nourish the human infant, yet, as already pointed out (p. 120) the insurmountable differences between the two varieties of milk make this not at all the necessary result. As a matter of fact, many variations from the standard are often required. It is important to understand the action of the different milk-elements in health and in the production of symptoms of indigestion, and with this knowledge, to change one or another until the suitable food is obtained. This action has already been studied to some extent in discussing the absorption and metabolism of breast-milk (pp. 48 and 52), and the action of the different elements of breast-milk (p. 99). It is rare, in my experience, that a healthy infant cannot be fed with reasonable success upon cow's milk, if it has been properly started. The problem arises in the case of those who have already been improperly fed. Occasionally, it is true, an idiosyncrasy to cow's milk is encountered, and such infants cannot take it

¹ Journ. Med. Research, 1912, XXVI, 127; Amer. Jour. Dis. Child., 1912, III, 226.

at all. This condition is generally an acquired one, dependent probably upon an anaphylaxis to cow's milk-protein developed through faulty efforts to feed this during a period of indigestion in very early infancy. The action of the ferments may be ignored in this connection, as we know comparatively little of the actual part played by them in the diet. The mutual influence which the different elements of the food exert upon each other is, however, a matter of importance and is receiving increasing attention.

Fat.—The fat of the milk-mixture is the element most easily causing digestive and nutritional disturbances, and many infants show a great and persistent intolerance for it. While in theory 4 per cent. should be well-borne, this being the amount existing in human milk, it is usually necessary, in my experience, to use decidedly less than this in making a milk-mixture, owing to the excess of volatile fatty acids present in cow's milk. This is particularly the case in very young healthy infants, and in others older whose digestion has been impaired by illness. For all such an initial percentage of 0.5 or 1.0 per cent. is to be selected, and the increase above this made very carefully. Even with those who have shown a power to digest cow's milk-fat, 3 per cent. or at most 3.5 per cent. is generally as high as it is safe to go. This is especially true in home-modifications if portions of top-milk and bottom-milk have been combined, since in the latter there is a certain amount of fat present which increases the percentage slightly above that indicated by the calculation. Fat is indeed the commonest cause of frequent sour vomiting with the rancid odor of butyric acid, although an excess of sugar may likewise produce sour, watery regurgitation. So, also, if the fat is in too large an amount it combines with the heavier alkalies, as calcium and magnesium, and produces soap-stools; or, uniting with potassium or sodium causes diarrhea with white, soft curds. The increase of the carbohydrates will sometimes cause the fat to be absorbed. Where there is an unusual intolerance for the fat of cow's milk, this element must be withdrawn, or reduced in amount. For this purpose butter-milk (p. 147) is suitable. Efforts have been made to use other kinds of fat, especially olive oil (Ladd).¹ It is necessary, however, to have this made into an extremely fine emulsion by a special process. Even lard and cocoanut oil have been tried (Gerstenberger, *et al.*).²

A very common practice with many physicians is the giving of a food-mixture rich in cream for the sake of overcoming constipation. This is a dangerous procedure in infants in the 1st year; and there are better ways. On the other hand, it is to be remembered that fat produces more than twice as many calories as do the other milk-elements, and that without a sufficient amount of it the quantity of food necessary to supply these may be unduly great. Infants who are not able to take a normal amount of fat are liable to be insufficiently nourished.

Carbohydrate.—The sugar of the milk is generally digested well. It is seldom necessary to begin with less than 5 per cent., and under ordinary circumstances it is not required to increase beyond 6 or 7 per cent. Much discussion has arisen as to the variety of sugar which should be employed. Usually it would seem best to select lactose for this purpose. This is the natural sugar of the food, and by its slower absorption tends to remain longer in the intestine, favoring the growth of the normal intestinal flora. It also undergoes butyric acid fermentation less readily

¹ Transac. Amer. Ped. Soc., 1915, XXVII, 117.

² Transac. Amer. Ped. Soc., 1915, XXVII, 94.

than does maltose, and is better tolerated than is this or cane-sugar when there is disordered gastric digestion with vomiting. On the other hand, under the influence of abnormal bacterial growth, lactose may become more harmful than other sugars. Where a rapid absorption and increase of weight is desired, saccharose and, especially, dextrin-maltose preparations are to be preferred. They are serviceable in this way when on account of difficulty in the digestion of fat it is necessary to supply the requisite calories by giving a high carbohydrate diet.

The carbohydrate in the food tends to favor the digestion of the protein, and when administered in high percentage will often change completely the character of stools showing protein indigestion. It often favors, too, the absorption of the fat; perhaps by increasing the acidity of the intestinal tract and thus preventing the union of the fatty acids with the alkalies to form soap-stools. If, however, given in excess, any form of sugar may cause diarrhea, and a consequent diminished absorption of the food-elements. Not more than 7 per cent. of lactose should be administered (Morse and Talbot),¹ and when larger percentages of carbohydrate are required, a dextrin-maltose preparation or starch is better.

The term "maltose" as used in the feeding of infants is generally employed to designate one of the combinations of dextrin and maltose on the market. There are a number well known, such as Soxhlet's Nährzucker, the malt-soup extracts, neutral maltose, and others and all may be denominated here "dextrin-maltose preparations." They consist of combinations of maltose and of dextrin, produced in the dextrinizing of starch. (See p. 155.) Similar preparations may be made at home. The percentage of maltose ranges roughly from 40 to 60 per cent. and of the dextrin from 10 to 60 per cent. The preparations containing the more maltose are more rapidly absorbed, but on the other hand are more liable to produce diarrhea, and those with considerable dextrin are to be preferred. The following table of analyses is in part that given by Southworth,² with the addition of figures for the Maltine Company's,³ Borchardt's⁴ and the Freihofer Company's⁵ products. Some of these

TABLE 43.—MALTOSE AND DEXTRIN OF MALT-EXTRACTS

	Maltose, per cent.	Dextrin, per cent.
Soxhlet's Nährzucker.....	52.44	41.21
Loeflund's Nährmaltose.....	40.00	60.00
Dextrin-maltose (Mead-Johnson).....	51.00	47.00
Neutral Maltose (Maltzyme Co.).....	63.00-66.00	8.0-9.0
Loeflund's Malt-soup Extract.....	58.91	15.42
Borchardt's Malt-soup Extract.....	57.57	11.70
Maltine Co. Malt-soup Extract.....	62.3	3.003
Freihofer Co. Malt Extract.....	61.0	6.0

preparations are distinctly acid, and the addition of from 5 to 10 grains (0.32 to 0.65) of potassium carbonate to the ounce is advisable. (See also p. 156.)

Many disordered conditions of the digestion have been attributed to the influence of the sugar of the food, and without question with more or

¹ Diseases of Nutrition and Infant Feeding, 1915, 264.

² Arch. of Ped., 1912, XXIX, 652.

³ Advertisement.

⁴ Advertisement.

⁵ Information received from manufacturer.

less reason. Yet a careful review of the subject by Porter and Dunn,¹ combined with their own experiments, led to the conclusion that the danger of severe symptoms from the giving of the sugars in considerable amount has been greatly exaggerated.

Starch.—It very frequently is of great advantage to employ the addition of starch in the food-mixture. Its caloric value is practically the same as that of sugar. Although the pancreatic secretion possesses amylolytic power even at birth, this is only slight before the age of 2 or 3 months, and when starch is given before this age it is for purposes other than absorption, such, for instance, as the prevention of the formation of curds. In the form of barley-water or other amylaceous decoctions it is often of great service. There is no reason, however, to give starch to every infant as a routine measure. In fact it should be used only for a definite purpose and in definite percentage-strength. Generally 1 per cent. at most is sufficient for simple dilutions. A table of the strength of various cereal decoctions will be given later (p. 154).

Starch is an extremely useful carbohydrate in those cases where a high percentage of this latter is required, being often better tolerated when combined with a dextrin-maltose preparation than is the latter when given alone. It is the existence of this combination which makes the malt-soup preparations so serviceable in many instances (p. 156). This is probably in part due to the mechanical colloidal action of the starch upon the protein, and in part dependent upon the slower breaking up of the starch-molecule as compared with sugar and dextrin. If, however, starch is given continuously in excessive amounts, and especially if without the addition of milk, or with only small amounts of it, severe symptoms may arise. The infant becomes pale, flabby and perhaps too plump; indigestion with diarrhea develops; there is undue fermentation in the alimentary tract, with loss of power of digestion and the development of a wasted condition, or sometimes of increase of weight dependent in part upon an extensive edema. (See Injury from Starch, p. 616.)

Protein.—Other things being equal, the protein of milk, and especially the casein, is tolerated in relatively high amount, and seldom produces disturbances of digestion. Yet it is better to begin with not over 1 per cent. in the mixture, and then gradually increase this as the needs demand. The quantity required by the infant is not very large, although greater relatively than in the adult. The average minimum amount is 1.5 grams per kilogram of body-weight (10.5 grains per pound), and many infants need slightly more than this (p. 50). The great difference between the protein and the other elements is that whereas these may replace each other to some extent this amount of protein is absolutely required, and can be substituted by nothing else. Infants fed upon a diet insufficient in protein cannot really thrive. Although they may often be quite fat, perhaps from the large amount of carbohydrate which has been administered, they are anemic, flabby and possess little actual strength or muscular development. As regards the different proteins of the milk, it has been claimed in many quarters that the casein is always tolerated in high percentage provided that of the whey is reduced; the benefit from the reduction depending probably upon the diminution in the amounts of sugar and of salts rather than of the lactalbumin. This requires further confirmation, and there is decided evidence opposed to it. It would seem probable, for instance, that the improvement in symptoms undoubtedly

¹ Amer. Jour. Dis. Child., 1915, X, 77.

sometimes seen after the employment of peptonizing, once so much in vogue, would indicate that the percentage of casein was not the matter of indifference which has been maintained. There is further no question from the experience of many physicians that many ill infants will thrive on whey-mixtures who have done badly upon food containing considerable casein. This would seem reasonably to be expected when one considers the comparatively large amount of whey-protein in human milk as compared with the casein. (See table 42, p. 119.) For many years whey-cream mixtures were popular with physicians, and deservedly so in selected cases. In fact, it has been pointed out (Holt)¹ that sometimes the feeding with high casein-percentages may not be tolerated unless whey, with its comparatively high carbohydrate content is present also; and that, further, the whey is needed on account of the large percentage of certain amino-acids present in it. Sometimes the whey in the mixture is well borne if the fat-percentage is low, as is the case when buttermilk is employed.

Sometimes the proteid-matter of milk is found in the stools as hard, yellowish-white curds (see p. 50), but this appears to be of little moment unless the curds are very numerous, and seems to be prevented if the milk has been heated to a high temperature, or by other measures already discussed (p. 124). Failure to digest protein also often results in the development of offensive, green, diarrheal stools, or of soap-stools with an offensive odor.

Much has been written concerning the digestibility and absorption of foreign proteins and the deleterious effects which may or may not occur. The subject has already been alluded to. (See p. 50.) There seems to be no question that such results can follow. In the cases described by Benjamin² there was pallor, great atony, free perspiration and sometimes eczema. In an instance reported by Hoobler³ a decidedly stuporous condition developed, whereas in cases reported by Holt and Levene⁴ there was continued fever. The influence of foreign proteins in the food even when in small amount may sometimes show itself in other ways. Thus, for instance, in the early weeks of life, and especially in infants suffering from any gastro-intestinal or nutritional disorder, it is possible for the foreign protein to pass unaltered or partly digested through the intestinal wall and to produce modified evidences of anaphylaxis. In some instances this amounts to an actual idiosyncrasy, and certain foreign proteins, even in very small quantity, cause decided and at times alarming symptoms, even in apparently healthy infants and in older children. This may occur with the casein of cow's milk, but an actual anaphylactic condition of this nature produced by it is certainly uncommon. Oftener it is seen in older children, and is especially noticed with the protein of egg or of certain of the cereals, varying with the case. Very similar symptoms can sometimes be occasioned by other food-stuffs, such as certain fruits. The symptoms consist not only in severe gastro-intestinal manifestations, but in nervous disturbances including prostration, cutaneous eruptions, dyspnea, sneezing, wheezing respiration, and other symptoms suggestive of an asthmatic attack. Cutaneous tests upon children have been made with the various proteins by Schloss,⁵ Talbot,⁶ and others, which confirm the clinical experience that it is the protein which is the active cause of

¹ Arch. of Ped., 1916, XXXIII, 13.

² Zeitschr. f. Kinderh., Orig., 1914, X, 183.

³ Amer. Jour. Dis. Child., 1915, X, 153.

⁴ Amer. Jour. Dis. Child., 1912, IV, 266.

⁵ Amer. Jour. Dis. Child., 1912, III, 341.

⁶ Bost. Med. and Surg. Journ., 1918, CLXXIX, 285.

the disturbance. Various protein-preparations to be used for cutaneous testing are now on the market commercially.

Mineral-matter.—The salts are certainly necessary for the life of the infant, and are absorbed and retained in proportion to the retention of nitrogen. With the exception of iron they are present in cow's milk to an excess which renders them sufficient for the infant given any ordinary dilution. It has been claimed that they are not always well tolerated, and that the reduction of the salts and of the sugar, as results from the removal or reduction of the whey, is of service in some cases of difficult digestion. The whole question of the part played by the salts in digestion is, however, far from being understood. (See also p. 51.)

The Selection of Percentages.—What follows may be regarded in the light of a summary of the substance of the preceding sections. At the outset it must be clearly stated that it is impossible to formulate any fixed rules for the percentage-strength of the milk-ingredients which even the normal child will require. The tables often published showing the strengths to be used at different ages are liable to be misleading and harmful, in that they tend to make the inexperienced practitioner attempt to fit the baby to the table, rather than the food to the baby. The fact has already been pointed out that after the early weeks the proportions of the ingredients of human milk do not alter materially. The infant simply takes a larger quantity as it grows older. In artificial feeding, on the other hand, owing to the difficulty in the digestion of the fat and the differences in composition between the proteins of cow's milk and of human milk, the percentage of these latter in the mixture usually must be raised, as otherwise the bulk of the food required to supply the protein needed might be far too great if properly diluted. The only dependable guide is the state of the general health of the infant, the condition of its digestion, and the rate of growth in weight. To make the age by months the guide is, I believe, a serious error. Should the health and weight be all that can be desired, increase in the strength of the food in any respect is to be made cautiously if at all. If the condition indicates an insufficient food-supply, the quantity is to be increased or the percentages raised. If digestive disturbances arise, the food must be further modified. There are naturally certain percentages which experience has taught that the average child can be expected to take at certain ages; but the feeding of each individual infant is a matter of trial, since no one can know in advance what the tolerance for cow's milk will prove to be.

With these limitations the following directions may be given as a general guide: The healthy infant in the first few weeks of life may be started with such a formula as fat 1 per cent., milk-sugar 5 per cent., and protein 1 per cent. Very probably there will be no gain in weight, or even a slight loss with this weak mixture, and it is likely that a stronger initial one could have been used. It is safer, however, to begin with the low formula in order to test the effects upon digestion. There is no hurry about the gain in weight. To accustom the infant to its new food and not to disturb the digestion are the important matters. If the food agrees well it may be changed in a few days to fat 1.5 per cent., and then again to fat 2 per cent., sugar 6 per cent., and protein 1.5 per cent. These changes, of course, go hand in hand with an increase in the quantity, which will be considered in the next section. With such a formula as the last given the infant may be quite contented and thrive well for a considerable time. Should, however, the digestion be in perfect

order but the infant hungry and the weight at a standstill, the strength must be increased to fat 2.5 or 3 per cent., sugar 6 or 7 per cent., and protein 1.5 or 2 per cent. It is manifest that hunger may be satisfied and gain in weight accomplished by giving an unduly large quantity of a weak formula, but the policy is not a good one. After the age of 6 months it is generally necessary to make a further increase in the strength of the food, giving perhaps fat 3 or 3.5 per cent., sugar 5 or 6 per cent., and protein 2 or 3 per cent. The frequent difficulty in digesting fat should still be borne in mind, and in most cases an amount over 3 per cent. should be tried with great caution. Many infants even at a year of age cannot take entirely undiluted cow's milk. What changes shall be made in the food, what elements increased or decreased in amount, is to be determined by a study of the effects, as outlined in the preceding section (p. 127). The method of preparing the food-mixture containing the percentages desired will be considered in the sections upon Laboratory-modification (p. 134) and Home-modification (p. 134) of the milk.

The Quantity of Food and the Frequency of Feeding.—The quantity of the milk-mixture to be given varies, of course, with the age, and approximates the table for the amount of breast-milk as given on p. 92. The frequency of feeding, too; the hours for this; and the relation of feeding to sleep, are governed by the principles which control breast-feeding (p. 84), and the following table, an elaborated form of that upon p. 85, gives an approximation of the frequency and the amounts for each feeding at different ages, the quantity being gradually increased as the age advances:

TABLE 44.—INTERVALS AND AMOUNTS FOR ARTIFICIAL FEEDING

Age	Intervals of feeding, hours	Number of feedings in 24 hours	Number of feedings at night after 10 P.M. and before 6 A.M.	Amount at each feeding, ounces	Total amount in 24 hours, ounces
1 to 4 weeks.....	2-2½	8-10	2	1-2	10-20
4 weeks to 3 months	2½-3	7-8	1	2½-5	20-21
3 to 4 or 5 months..	3-3½	6-7	1(?)	4-6	28-36
4 or 5 months to 1 year.	3-4	5-6	0	6-10	30-50

The length of the intervals and the number of feedings are much the same as for breast-feeding, and the divergence of opinions regarding the matter has been referred to in that section. There is, of course, much latitude to be allowed in using the table, dependent upon the digestion and the demands of the child. Regularity is important, but the amount taken at a feeding and the number of feedings in the twenty-four hours vary greatly with the case. An infant always hungry may need to exceed the amount for its age or to be fed at shorter intervals; another with large appetite may need longer intervals. It is important, too, to be guided considerably by the weight. An unusually large child may require more, while a small marantic infant may be able to take only much less than the age calls for. There can be no fixed rule. In general, a healthy infant may have all the food it will take at the feeding-time, provided it can retain and digest it; but care should be observed that an unusually large appetite is not dependent upon the giving of too weak a formula.

Laboratory=modification of Milk.—Through the efforts of¹ Dr. Rotch,¹ ably seconded by Mr. G. E. Gordon the principle of the "Milk Laboratory" was evolved, and one such established in Boston in 1891 by the Walker-Gordon Laboratory Company. Similar laboratories have since been organized in many of the larger cities of the United States and in London. These institutions have been of the greatest benefit in giving an impetus to the scientific feeding of infants. Their object is to prepare and furnish any percentage food-combination ordered by the physician, including those in which "differential protein" feeding is employed; *i.e.* in which the lactalbumin and casein are prescribed separately; and those in which cereal decoctions of known strength, buttermilk, and the like take a part. The physician merely writes a prescription calling for the various percentages he needs, the total number of bottles, the amount of food for each, the nature of the diluent, the alkalinity, and any other requirement desired. If the food is to be pasteurized or sterilized, a starchy addition to be dextrinized, acidulation by lactic acid bacilli to take place, or any other change made, this is specified in the prescription. It is manifest that a milk-laboratory is not only a great convenience to the physician to whom it is accessible, but that the resulting mixtures will be more accurately compounded than when made at home; since the employees are certainly better trained than is the mother or the nurse, and a careful control can be kept over the percentage-strength of the milk and cream used. Unfortunately the price of laboratory modified milk is necessarily considerably higher than persons of restricted means can afford, and for this reason, as well as on account of the usual inaccessibility of a laboratory for the majority of infants, home-modification is that which must be oftenest adopted.

The Home=modification of Milk.—The calculation by the physician of the desired formulæ is in reality a very simple process. The preparation of the food can be carried out by any ordinarily intelligent and properly directed mother or nurse. The chief advantage of home-modification consists in its economy as compared with laboratory feeding. Although less accurate than this latter, it approaches it closely if a cream of definite known strength can be procured; and even for home-prepared whole-milk or top-milk mixtures it is quite sufficiently accurate for ordinary purposes. This is especially true since the possibility has extended so widely of procuring milk certified by a recognized Milk Commission. Milk of this sort should be obtained whenever feasible; and, when not, the requirements described (p. 117) should be fulfilled as far as possible. It is particularly important that it be of a fairly uniform strength in fat, and not over 4 per cent. When it is necessary to employ the richer milk, a slight change in the procedure is required. (See pp. 117 and 139.)

Many unwarranted statements have been made and published regarding the difficulty supposed to attend the preparation of milk-mixtures in the home, and the complications involved in the prescribing of these by physicians. Percentage-feeding and home-modification may be made unnecessarily difficult, it is true, but do not in any way need to be so. The calculation of the desired percentages (p. 137) may, as already indicated, be made by any physician with the simplest school-boy knowledge of arithmetic. It is not so much lack of knowledge as unwillingness to take trouble which interferes on the part of the physician;

¹ Arch. of Ped., 1893, X, 97.

but to prescribe the best obtainable food for the baby is so important that there can be no possible excuse for any physician who will not undertake to do this in the best way. The objection that home-modification of milk is too complicated for the mother is so futile that it hardly merits consideration. The mother is obliged to modify the milk in some way, and it can certainly cause no more trouble to dilute, say, the upper half of a quart of milk with water, than to dilute the whole quart; nor is the addition of skimmed milk and water, or top-milk and water, to the mixture any more complicated than the adding of water alone. The simplest way is the best way; and if the desired results can be obtained by the dilution of whole milk with water, this may well be done. But in the very numerous instances where a mixture is required in which the percentage of the fat is greater than that of the protein, it is only by the employing of a top-milk that the results can be obtained. With the aid of clear, concise, written directions there is little chance of error; and mothers to whom the first preparation of any milk-mixture seems difficult rapidly find the processes becoming easy. But if the physician is hazy in his own mind regarding the matter, or neglects to give explicit instructions, the mother naturally grows confused and discouraged, whatever sort of food is ordered and whatever method employed.

Articles Required in Preparing the Food.—*Bottles.*—These should be of well annealed glass to prevent breaking when exposed to heat. They may be of any shape desired, but are best narrow and cylindrical, since they occupy less space when of this form, and fit better in the pasteurizer. The mouths of the bottles should be rather wide, rendering cleansing easier. The bottles must be perfectly smooth within, without angles or depressions which can collect milk. A series of markings pressed into the glass indicate the number of ounces of contained fluid. There should be enough bottles provided to permit of all the food for twenty-four hours being prepared at one time.

Nipples.—These are preferably of black rubber and of conical or slightly bulbous shape. The openings should be only large enough to allow the milk to drop easily when the filled bottle is inverted, but not to run from it in a stream. If the holes are not of sufficient size they may be enlarged with a red-hot needle. As the nipple gets worn by use it collapses too easily and the holes grow too large. Special forms of nipples to “ventilate” the bottle are not to be recommended, as it is difficult to keep them clean. Too long a nipple is liable to press upon the palate and produce ulceration. The nipple attached to a rubber tube cannot be sufficiently condemned, as it is almost impossible to keep the tube clean. It is fortunately largely out of vogue.

Cream-dipper.—For the purpose of obtaining top-milk the most satisfactory method is the employment of the Chapin dipper. This has already been referred to (p. 116).

Sugar-measure.—The most convenient method of measuring the lactose is to employ the Chapin dipper. While holding 1 oz. of milk, it gives slightly less than $\frac{1}{2}$ oz. (Av.) of milk-sugar. This fact must be impressed upon the mother. Another way is to employ a tablespoon, but this is less satisfactory, as the variation in size is so great. Approximately $3\frac{1}{4}$ level tablespoonfuls, 2 rounded tablespoonfuls or $1\frac{1}{2}$ dipperfuls of milk-sugar is equivalent to 1 oz. Av. Still another method is to place 1 oz. of sugar, as determined by weighing it, in a small paste board box, level it, and then cut off the box at the line of the sugar.

If tablespoonfuls are to be employed, the level one is better, inasmuch as the rounded tablespoonful gives more variation. Whatever method is used, the sugar should be poured into the measure until it is over-filled; the holder tapped sharply once or twice on the table, not more, and the excess of sugar removed with a case-knife. Too long tapping settles the sugar to too great an extent. Cane-sugar and the dextrin-maltose preparations vary somewhat in weight from that of lactose, the latter being a trifle lighter and the former decidedly heavier. Should it be desired to substitute one of them for lactose, it may be roughly estimated that 1 oz. of lactose equals $3\frac{1}{4}$ level tablespoonfuls or $1\frac{1}{2}$ dipperfuls; 1 oz. of a dextrin-maltose, 4 level tablespoonfuls or 2 dipperfuls; and 1 oz. of saccharose, $2\frac{1}{4}$ level tablespoonfuls or 1 dipperful. (For weight of tablespoonfuls and dipperfuls of sugar and cereal-flours see also p. 153.)

Glass Graduate; etc.—One of those holding 8 fl.oz., with the first ounce divided into drachms, is a great convenience in measuring the amounts of fluid required. One of the graduated nursing bottles may, it is true, be used instead, but these are not so accurately marked.

In addition are required a glass funnel, a bottle-brush, some sterilized unabsorbent cotton to make the stoppers for the bottles, pulverized boric acid, a bowl or pitcher in which to mix the food, and a Freeman pasteurizer described on p. 126.

Regarding the ingredients required, the best milk possible is to be obtained, running close to 4 per cent. butter-fat. (See *Certified Milk*, p. 118.) If purchased cream is used it should be separator cream, freshly obtained, of a guaranteed percentage-strength in fat, and with a low bacterial content. Milk-sugar should be purchased at least a pound at a time, as it is cheaper in this way. The fact that it is frequently impure is not to be forgotten. If lime-water is employed, it may be purchased or made at home. In the latter case a piece of unslaked lime the size of an egg is put into a gallon of water, which is then stirred vigorously and allowed to settle. The first water is poured off and fresh added. It should be kept covered.

Preparing and Giving the Food.—First is determined the number of bottles which the infant shall have in twenty-four hours and the number of ounces at each feeding, the multiplication of one number by the other giving, of course, the total amount required for the day. All of this is to be prepared at the one time, as soon as possible after the milk arrives. Should delay be unavoidable the milk must be kept on ice until needed. The required amount of sugar is then measured in the manner described, added to the amount of water or other diluent needed for the mixture, this raised to the boiling point for a moment, and then cooled. As the addition of the sugar increases the volume of the water slightly, greater accuracy may be obtained by boiling the sugar in a *portion* of the water only, and then adding enough additional water to bring the volume up to that of the total amount of solution to be added. The difference, however, is not very considerable. The boiling of the sugar-solution is necessary only if the mixture is to be used raw, on account of the bacterial content of the sugar often existing.

The amount of top-milk, cream, whole-milk, skimmed milk, and water—as the case may be—is now measured and added to the sugar-solution and all the ingredients mixed well, the volume of the whole equalling the amount of food desired for twenty-four hours. Should it be intended to sterilize the mixture at 212°F . (100°C .) and the use of lime-water be desired, this should not be added until the process is over. With pas-

teurizing at 140° to 150°F . (60° to 65.5°C .) there is no such necessity, inasmuch as the lime-water is not affected by this temperature. Into each bottle is now poured the amount for each feeding, and the neck of the bottle wiped clean and dry and then stoppered with a cotton-plug. The bottles are now put into the pasteurizer and the food pasteurized, if this is desired, cooled, and kept on ice. When a bottle of food is needed, and then only, it is removed from the ice, and placed in a tin of cool water; and this is then warmed until the milk is at 98 to 100°F . (36.7° to 37.8°C .), but not more. This is determined by a thermometer made for the purpose and placed in the milk-bottle, not in the water (Fig. 22). If the bottle is plunged directly into hot water the glass is liable to crack. The nipple is now put on and the child fed. In heating the food a tin sufficiently tall should be employed to allow the water to cover the bottles to the neck, but narrow enough to reduce to a minimum the actual amount of water required (Fig. 22). Placed upon a small alcohol heater or gas-stove, the water and bottle of milk will thus be raised quite rapidly to the required temperature. This rapidity is an important matter when a hungry child is crying impatiently for food.

As with breast-feeding, regularity in the hours for food must be followed. The child should empty its bottle in from 10 to 20 minutes, and should not be allowed to suck at it indefinitely or to go to sleep until it has finished its meal. Any milk left over must be thrown away at once; never warmed up and given again later. The bottle should be held by the hand so that its neck is always full of milk. It should be drawn from the mouth from time to time to allow air to enter through the nipple in order to keep this from collapsing. The child should be held in the lap or in the arms while being fed. It is a bad plan to prop the bottle up with pillows to enable the infant to suck while it is in bed. After being used the bottle should be at once washed with a strong solution of washing soda, scrubbed with a bottle-brush, and rinsed out well with pure water. Shortly before they are filled all the bottles should be boiled at one time. The nipples should be scrubbed thoroughly without and, after turning inside out, within, and afterward be kept submerged in water or in a borie acid solution until needed. Before using it is well to plunge them for a moment in boiling water.

The Calculation of the Milk-formulæ.—In employing home-modification, milk-mixtures may be made either (1) from a combination of whole-milk and cream of a definite known strength, or (2) from dilutions of whole milk, top-milk or skimmed milk. The pages which follow are for the guidance of those readers prepared to take for themselves an active interest in the calculation of the amounts of the different elements required in the food. For those who would prefer to avoid the trouble, the table given on p. 142 may be found a useful guide. Various methods of computing the percentages have been proposed; none of them giving other than approximately accurate results, but near enough for practical purposes.

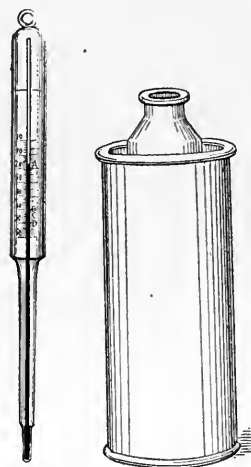


FIG. 22.—MILK-BOTTLE, HEATING TIN, AND THERMOMETER.

1. Whole-milk-and-cream Mixtures.—This was almost the only method employed for home-modification some years ago, but it has since given place almost entirely to top-milk mixtures. It is explained here because still in use by some physicians or in some hospitals; and because it has distinct advantages in occasional instances. It is applicable only when a cream of definitely known strength can be obtained. The simplest method of determining the amount of whole-milk and of cream required is that proposed by Baner.¹ We need not go into the derivation of his equations, but the calculation is as follows:

Let Q = the total quantity of food needed for 24 hours.
 F = the percentage of fat desired in the mixture.
 P = the percentage of protein desired in the mixture.
 S = the percentage of sugar desired in the mixture.
 C = the amount of cream to be used in the mixture.
 CF = the percentage-strength of the cream in fat.
 L = the amount of sugar (lactose) to be added to the mixture.
 W = the amount of water to be added to the mixture.

Then (I) $C = \frac{Q}{CF - 4} \times (F - P).$

(II) $M = \frac{Q \times P}{4} - C.$

(III) $W = Q - (M + C).$

(IV) $L = \frac{Q \times (S - P)}{100}.$

The denominator of equation (I) is always the percentage-strength of the cream in fat, minus 4.

As an example of the use of the formulæ, suppose it is desired to give an infant 8 bottles of 4 oz. each, making a daily quantity of 32 oz.; that this shall contain fat 3 per cent., sugar 6 per cent. and protein 1.5 per cent., and that the cream is of 20 per cent. fat-strength.

If we substitute these figures in the equations these read:

(I) $C = \frac{32 \times (3 - 1.50)}{20 - 4} = \frac{32 \times 1.5}{16} = 3; \text{ i.e. } C = 3 \text{ oz. 20 per cent. cream.}$

(II) $M = \frac{32 \times 1.50}{4} - 3 = 9; \text{ i.e. } M = 9 \text{ oz. whole milk.}$

(III) $W = 32 - (9 + 3) = 20 \text{ oz. water.}$

(IV) $L = \frac{32 \times (6 - 1.50)}{100} = 1.44; \text{ i.e. practically 1.5 oz. sugar.}$

Should Equation II result in 0, it would mean that no milk, but only cream of the strength indicated, is to be used in making the mixture. Resulting in a minus quantity it denotes that the formula desired cannot be constructed with the strength of cream employed, and that one with a higher percentage of fat must be selected. When it is necessary to employ a milk containing 5 per cent. of fat, the figures may be reduced to 4 per cent. by dipping off and discarding the top 2 ounces of the quart after the cream has separated, and then shaking up the bottle well before the remaining milk is used as "whole milk."

The criticism may properly be made that Baner's formulæ are inaccurate, since they assume that milk and cream each contain 4 per cent. of protein, whereas milk more often has 3.5 per cent., and cream has some-

¹ New York Med. Jour., 1898, Mar. 12, 345.

what less sugar and protein than has whole milk. A comparison of the real and the calculated protein-percentages obtained shows, however, differences which are entirely negligible. It is only when employing milk-mixtures containing as much as 3 per cent. of protein that the difference between the calculated and the actual protein amounts present reaches as much as somewhat less than 0.5 per cent.

2. Top-milk, Skimmed-milk and Whole-milk mixtures.—In recent years the dilution of top-milk, whole-milk or skimmed-milk has become the popular method of preparing the food. It offers every advantage in convenience and in reasonably approximate accuracy in the percentages present. Many figures have been given by different investigators of the strengths of varying numbers of ounces of the milk in a quart milk-jar. They are close enough to show that this method of feeding can be employed satisfactorily. With the exception of the fat, the average infant can, as a rule, tolerate decided variations in the milk-elements; and the important matter after all, as has been stated previously, is not so much the initial accuracy of the percentages as it is the comparative accuracy of the *changes* in the food which it may be desired to make from time to time.

With this in mind, and aware especially of the importance of rendering the calculating as simple and as comprehensible as possible, I have for a number of years followed and taught the following plan. The percentages are based upon the published figures of different observers and upon unpublished investigations made at my suggestion by Dr. Charles A. Fife. The percentages of protein and of sugar have intentionally been slightly distorted for reasons presently to be given. The title "skimmed-milk" designates any milk from which all or a portion of the cream has been removed. (See p. 117.)

TABLE 45.—PERCENTAGE-STRENGTHS OF TOP-MILK AND SKIMMED-MILK LAYERS

Milk obtained from a quart of 4 per cent. milk by dipping, as previously described (p. 116), gives:

	Fat, per cent.	Protein and sugar, per cent.	Approximate ratio
Upper 2 oz.....	24.0	4	.6 to 1
Upper 4 oz.....	20.0	4	.5 to 1
Upper 6 oz.....	16.0	4	.4 to 1
Upper 8 oz.....	12.0	4	.3 to 1
Upper 10 oz.....	10.0	4	2.5 to 1
Upper 16 oz.....	8.0	4	.2 to 1
Upper 20 oz.....	6.0	4	1.5 to 1
Upper 24 oz.....	5.0	4	1.25 to 1
Upper 32 oz. (whole-milk).....	4.0	4	.1 to 1
Lower 30 oz.....	3.0	4	0.75 to 1
Lower 28 oz.....	2.0	4	0.50 to 1
Lower 16 oz.....	1.0	4	0.25 to 1
Lower 8 oz.....	0.5	4	0.0 to 1 ¹

This table applies to milk containing approximately 4 per cent. of fat. With rich milk, showing 5 per cent. of fat, all that is necessary is to remove and discard the top 2 oz. and then to proceed with the dipping as though this had not been done. That is to say, by the "top 8 oz." for example, is indicated the upper 8 oz. remaining after the top 2 oz. have been dis-

¹The amount of fat in the lower 8 oz. is so small that it may be ignored in the "approximate ratio."

carded. The figures in the table are not absolutely accurate, and are not intended to be. They are sufficiently so for practical purposes, and the discrepancy is slight as compared with the great convenience in application. As previously shown, the protein of whole milk is nearer 3.5 than 4 per cent., and is even slightly less than this in the top-milk usually employed; but the difference after the required dilution has taken place is so slight that it is negligible. This statement applies equally well to the difference between the adopted 4 per cent. of sugar and the actual 4.5 per cent., and to the fact that the upper 16 oz. is more properly stated as containing from 7 to 7.5 per cent. fat. The great convenience of the approximate ratios which the table shows quite justifies the slight distortion of the figures. It is only when high protein-percentages are used that the discrepancy in the percentages is to be considered—a calculated protein-strength of 3 per cent. in the mixture showing a deficiency of not quite 0.5 per cent. If one bears in mind the protein-needs of the infant (pp. 50 and 130) it is easy to determine whether the deficiency is one which must be considered. This will not be necessary unless the minimum requirement is being closely approached, and this, of course, will not happen when high protein mixtures are being employed.

In employing the table we can conveniently follow the common custom of making a 20-oz. mixture our basis. Should we need, for instance, 36 oz., twice the 20 oz. may be prepared, and the extra 4 ounces discarded; if 30 oz. is required, $1\frac{1}{2}$ times the 20 oz. is made. It is to be noted first of all that the percentages of protein and of sugar are the same. Whatever percentage we deduce for the former will be equalled by the latter. Our first step is to determine what percentages of fat, sugar and protein are wanted in the milk-mixture. Suppose, for instance, that we desire one of fat 3 per cent., sugar 6 per cent. and protein 1 per cent. The ratio of fat to protein is that of 3 to 1. In consulting the table it will be seen that the upper 8 oz. containing 12 per cent. of fat possesses this ratio, and is the proper one to use. Now it is evident that if 20 oz. of milk contain 12 per cent. of fat, and we wish to reduce it to 3 per cent., *i.e.*, $\frac{1}{4}$, we merely divide the 20 by 4, and make up the rest with water, *i.e.*, we use 5 oz. of the upper 8 oz., and 15 oz. of water. This may be expressed in an equation, to which reference will be made from time to time as the *basic equation*.

BASIC EQUATION TO OBTAIN THE DESIRED FAT-PERCENTAGE

$$\frac{\text{Total quantity of food} \times \text{Percentage of fat desired}}{\text{Fat-strength of the milk used}} = \frac{\text{Quantity of top-milk or skimmed-milk in the mixture.}}$$

Applying this to the milk-mixture in question the equation reads:

$$\frac{20 \times 3}{12} = 5 \text{ oz. of upper 8 oz. in a 20 oz. mixture.}$$

The calculation for the dilution to obtain the fat-percentage is *the only one required*, for the desired dilution of the protein follows automatically, and the original 4 per cent. is reduced to 1 per cent. The sugar has been cut down to the same amount, since the percentages of the two are always the same. Wishing however, to have the sugar in the mixture 6 per cent., it is clear that since 1 per cent. is already present, we need only to add 5 per cent.; *i.e.* $20 \times 0.05 = 1$ ounce of sugar to be added to the 20 oz. mixture. Our milk-formula will then read:

Upper 8 oz., 5 oz.
Milk-sugar, 1 oz.
Water to make 20 oz.

Very frequently a weak cereal-decoction is employed as a diluent. This naturally increases the total carbohydrate-percentage in the mixture; but as a rule this may be ignored, and account taken of the sugar only. Should, however, one wish to allow for the difference, and to add, say, 1 per cent. of starch, reducing the sugar-addition to 4 per cent., we may make a decoction of 1.80 per cent. of starch (see p. 154), and again apply the equation just given, the starch replacing the fat in it. This reads: $\frac{20 \times 1}{1.80}$ = approximately 11 oz., indicating that this amount of the cereal decoction is to be used in place of an equal number of ounces of water to make a 20 oz. mixture. The formula would then read:

Upper 8 oz., 5 oz.
Sugar, 0.8 oz. (*i.e.* 4 per cent. of 20 oz.).
Barley-water, 11 oz.
Water to make 20 oz.

Generally, however, such a refinement of calculation is not necessary.

One more example may be given. Let us suppose that we desire to make the protein-percentage in the mixture given 2 per cent. instead of 1 per cent.; viz. fat 3 per cent., sugar 6 per cent., protein 2 per cent.—a ratio of 3 to 2, with the sugar 6 per cent. as before. Two courses are open to us. First, and most simply, we can select the upper 20 oz. of the quart, containing 6 per cent. of fat and 4 per cent. of sugar—a ratio of 3 to 2, or $1\frac{1}{2}$ to 1, as shown in the table. Using the same basic equation to obtain the percentage of fat, we have $\frac{20 \times 3}{6}$ = 10 oz. of the top 20 oz. of the quart required in the 20 oz. mixture. The proportions of protein and of sugar have automatically become 2 per cent., the ratio of 3 to 2 holding good. The other course is as follows: Using, for instance, the upper 8 oz. as in the first example we derive the mixture:

Upper 8 oz., 5.
Water, 15.,

giving fat 3 per cent., protein 1 per cent. To increase the protein to 2 per cent. without affecting the fat, it is only necessary to add a sufficient amount of milk in which the fat is a negligible quantity, as in the bottom 8 oz. of the quart. If this contains 4 per cent. of protein, it is evident that $\frac{1}{4}$ of the 20 oz. would contain 1 per cent. of protein. That is to say, by adding 5 oz. of skimmed-milk to the mixture in place of 5 oz. of water we shall have increased the protein-percentage from 1 per cent. to 2 per cent. Our milk-formula would then read:

Upper 8 oz., 5 oz.
Lower 8 oz., 5 oz.
Water, 10 oz.

Having 2 per cent. of protein in the mixture we necessarily have 2 per cent. of sugar, and all that is required to obtain the desired 6 per cent. of sugar is to add the additional 4 per cent.; *i.e.* 20×0.04 = 0.8 oz.

If a quantity of food is required which calls, for instance, for 10 oz. of the upper 8 oz., it is, of course, necessary to take 8 oz. from each of 2 quarts, or 8 oz. from a quart and 4 oz. from a pint, mix them and use 10 oz. of the mixture.

Where mixtures are desired in which the protein-percentage is in excess of the fat-percentage; *i.e.* skimmed milk mixtures, the method

of procedure is, of course, the same, using only the lower layers in the table in place of the upper. Thus for a mixture of fat 1 per cent. and protein 2 per cent., we can employ the lower 28 oz., containing 2 per cent. of fat, and having the ratio of 0.5 to 1. Our equation for the fat gives us $\frac{20 \times 1}{2} = 10$ oz. of the lower 28 oz. required in a 20 oz. mixture, this naturally reducing the 2 per cent. of fat and 4 per cent. of protein to 1 per cent. of fat and 2 per cent. of protein respectively.

In the cases where it is desired that the percentages of fat and of protein shall be approximately equal, whole-milk may well be employed. If, for instance, a formula of fat 2.5 per cent., sugar 5 per cent., and pro-

Ready Method for Selecting Amounts to be Employed in Making Various 20-Oz. Milk-Mixtures, and the Caloric Values Resulting															
Percentages desired of			Lower 8 oz.	Low- er 16 oz.	Low- er 28 oz.	Whole Milk	Up- per 24 oz.	Up- per 20 oz.	Up- per 16 oz.	Up- per 10 oz.	Up- per 8 oz.	Water oz.	Sugar oz.	Caloric Value of Mixture	Calories per oz.
Fat	Sugar	Prot'n													
0.5	5	1	5	15	0.8	175	8.75
0.5	6	2	..	10	10	0.8	225	11.25
1	6	1	5	15	1	225	11.25
1	6	1.5	2.5	5	12.5	0.9	237.5	11.88
1	6	2	10	10	0.8	250	12.5
1.5	6	1	5	15	1	250	12.5
1.5	6	1.5	7.5	12.5	0.9	262.5	13.13
2	6	1.5	2.5	5	12.5	0.9	287.5	14.38
2	6	2	10	10	0.8	300	15
2.5	6	1.5	2.5	5	12.5	0.9	312.5	15.63
2.5	6	2	10	10	0.8	325	16.25
2.5	6	2.5	12.5	7.5	0.7	337.5	16.88
3	6	1	5	..	15	1	325	16.25
3	6	1.5	2.5	5	..	12.5	0.9	337.5	16.88
3	6	2	10	10	0.8	350	17.5
3	6	3	15	5	0.4	375	18.75
4	4	4	20	0	..	400	20

FIG. 23.—AUTHOR'S MILK-CARD.

Showing amount of different layers to be used for the preparation of various percentage-mixtures, and the caloric values of the mixtures. (*Griffith, Journ. Amer. Med. Assoc.*, 1918, LXXI, 441.)

tein 2.5 per cent. is desired, we substitute in the basic equation the percentage values of whole-milk as given in the table (p. 139) as follows: $\frac{20 \times 2.5}{4} = 12.5$ ounces whole-milk in the 20 oz. mixture. As this gives

us necessarily also 2.5 per cent. of protein and 2.5 per cent. of sugar, the addition of 2.5 per cent. of sugar is obtained as before; i.e. $20 \times 0.025 = 0.5$ oz. of sugar to be added.

The whole matter of calculation will be found extremely simple upon trial. It may be summarized in the following three rules:

1. *Select the top, skimmed or whole milk which possesses the ratio of fat to protein desired for the mixture* (Table 45, p. 139).

2. *By using the equation on p. 140, calculate the amount of this milk needed in a 20 oz. mixture to give the desired fat-percentage. The results will give the desired protein-percentage also.*

3. *The sugar having been, of course, reduced equally with the protein, add as much more to the 20 oz. as is needed to raise it to the desired percentage.*

The figures in the table (p. 139) run so easily in sequence that they are carried in mind without effort, and a little consideration will reveal many possibilities of producing the same results by different methods. Thus a formula, calling for 3 per cent. of fat and 2 per cent. of protein can be made, as already shown (p. 141), either by diluting 10 oz. of the upper 20 oz. of the quart with 10 oz. of water, or by mixing 5 oz. of the top 8 oz. with 5 oz. of the bottom, and adding 10 oz. of water. One requiring 2 per cent. of fat and 1 per cent. of protein may consist of 5 oz. of the top 16 oz. of the quart and 15 oz. of water; or of 3½ oz. of the top 8 oz., 1½ oz. of the bottom, and 15 oz. of water. One calling for 2 per cent. of fat and 2 per cent. of protein may be constructed simply by diluting whole-milk with an equal amount of water, or by mixing 4 oz. of the top 10 oz. with 6 oz. of the bottom 8 oz. and 10 oz. of water. In each case the

Table Giving Approximate Percentage-Strengths of Different Layers of Milk

	Per cent. Fat	Per cent. Protein and Sugar	Ratio
Upper 2 oz.....	24	4	6 to 1
" 4 ".....	20	4	5 to 1
" 6 ".....	16	4	4 to 1
" 8 ".....	12	4	3 to 1
" 10 ".....	10	4	2.5 to 1
" 16 ".....	8	4	2 to 1
" 20 ".....	6	4	1.5 to 1
" 24 ".....	5	4	1.25 to 1
" 32 " { whole milk }	4	4	1 to 1
Lower 30 ".....	3	4	.75 to 1
" 28 ".....	2	4	.50 to 1
" 16 ".....	1	4	.25 to 1
" 8 ".....	0.5	4	.0 to 1

To Find the Amount of Any Layer of Milk to be Used to Give Percentages Desired

Equation: -
$$\frac{\text{Total amount of food} \times \text{Percentage of fat desired}}{\text{Fat-strength of layer of milk used}} = \text{Amount of this milk in the mixture.}$$

(1) Select from the "Layers of Milk" Table the milk which possesses the desired ratio of fat to protein.
(2) Substitute in the equation.
(3) As the sugar-percentage has been reduced equally with that of the protein, add sufficient sugar to raise to the desired percentage.
EXAMPLE: 20-oz. mixture desired. Percentages desired = Fat 3, Sugar 6, Protein 1. Use upper 8 oz. (fat 12%, protein 4%, viz: 3 : 1). Then $\frac{20 \times 3}{12} = 5$ oz. of upper 8 oz., with 15 oz. of water in the 20-oz. mixture. The protein necessarily becomes 1%, and the sugar likewise. The mixture already containing 1% of sugar, add 5% of 20 oz., i.e., 1 oz. of sugar to increase this to the 6% desired.

To Determine the Percentages Present in Any Milk-Mixture Already in Use

$$\frac{\text{Quantity of substance used (milk, cream, or skimmed milk)} \times \text{Its percentage-strength}}{\text{Total Quantity of Food}} = \frac{\text{Percentage of element (F., S., or P.) in the mixture.}}$$

EXAMPLE: The mother has mixed: Upper 8 oz.; 6 oz.—Lower 8 oz.; 3 oz.—Milk-sugar 3 level tablespoonfuls.—Water 27 oz. Total quantity = 36 oz. The upper 8 oz. contains 12% fat (see Table). Both top and bottom milk contain 4% protein and sugar. Three tablespoonfuls sugar = approximately 1 oz. The fat of the lower 8 oz. may be ignored. Then $\frac{6 \times 12}{36} = 2$ = Fat percentage from the top-milk. $\frac{3 \times 0}{36} = 0$ = Fat-percentage from the bottom milk. $\frac{9 \times 4}{36} = 1$ = Protein and sugar percentages from combined top and bottom milk. The 1 oz. additional sugar divided by 36 = approximately 3% sugar added. There being already 1% sugar derived from the milk, the total sugar = 4%.

FIG. 24.—AUTHOR'S MILK-CARD.

Reverse side of Fig. 23, showing the percentages in different layers of milk; the calculation of the amount to be used for any formula desired, and the calculation of the percentages in any mixture already in use. (*Griffith, Journ. Amer. Med. Assoc.*, 1918, LXXI, 441.)

requisite percentage of sugar must be added. The simplest way is usually the best, both for the physician and for the mother or nurse.

For ready reference, those who desire to avoid all calculation as far as possible may employ the table shown in Fig. 23. This is based on the percentage-strengths as given on p. 139. Fraley's formula has been used for the determination of the caloric values. (See p. 123.) The milk-mixtures selected are those most likely to be found useful. The employment of fractional percentages smaller than those given offers no practical advantage.

To Ascertain the Percentages Present in any Milk-mixture.—Inasmuch as the description of the food in use is always given by the mother in terms of milk, cream, top-milk, water, etc., it is important to be able readily to ascertain what percentages of the milk-elements the child is actually receiving, in order to determine whether any ingredient is

manifestly present in incorrect proportion and is probably at fault if symptoms of indigestion exist.

The procedure is simple. To calculate the total percentage of any of the milk-elements present in the mixture, multiply the quantity of the substance (milk, cream, etc.) containing it by the percentage of the element present in this, and divide by the total number of ounces of the mixture. In the form of an equation it would read:

$$\frac{\text{Quantity of substance used (milk, cream, etc.)} \times \text{Its percentage-strength}}{\text{Total quantity of food}} = \text{Percentage of ingredient in the mixture.}$$

This is employed to determine both the fat-percentage and that of the protein and sugar. Supposing, for instance, we are told that the infant has been receiving a mixture of:

Upper 8 oz. of a quart, 6 oz.
Lower 8 oz. of a quart, 3 oz.
Sugar, 3 rounded tablespoonfuls.
Barley-water, 27 oz.

making a total food-mixture of 36 oz., containing a total of 9 oz. of milk in all. We are told that the barley-water is made by cooking 2 level tablespoonfuls of barley in 1 quart of water, and replacing the water which has been boiled away. Using now the Table of Percentage Strengths, p. 139, with the same restrictions as to accuracy as explained regarding it, we have in the upper 8 oz., fat 12 per cent., protein and sugar each 4 per cent.; in the lower 8 oz., fat practically 0 per cent., protein and sugar each 4 per cent. The 3 rounded tablespoonfuls of sugar are equivalent to $1\frac{1}{2}$ oz. (pp. 135, 153). The barley-water gives a percentage of 1.20 of starch (see table 51, p. 154). Substituting these various values in the equation the figures are as follows:

$$\begin{array}{ll} \frac{6 \times 12}{36} = 2 & \text{Fat per cent. obtained from the top-milk.} \\ \frac{3 \times 0}{36} = 0 & \text{Fat per cent. obtained from the bottom-milk.} \\ \frac{9 \times 4}{36} = 1 & \text{Per cent. of protein obtained from the combined top- and bottom-milks (6 + 3).} \\ \frac{9 \times 4}{36} = 1 & \text{Per cent. of carbohydrate obtained from the combined top- and bottom-milks.} \\ \frac{27 \times 1.20}{36} = 0.9 & \text{Per cent. of carbohydrate obtained from the barley-water.} \end{array}$$

Besides this we have added 1.5 oz. of extra sugar to the 36 oz. of mixture; and dividing the 1.5 by 36 this furnishes a percentage-addition of approximately 4 per cent., which, with the sugar already present, and the carbohydrate of the barley-water, gives a total carbohydrate-percentage of about 6 per cent. The food is thus found to contain fat 2 per cent.; carbohydrates 6 per cent.; protein 1 per cent.

For the convenience of my students I had the table for the ready method of the preparation of milk-mixtures printed in pocket-card form (Fig. 23), and upon the reverse side (Fig. 24), the table for layer-milk (p. 139) and the various matters relating to the calculation of formulæ as already described.¹

¹ These cards may be obtained from Edward Pennoek, 3609 Woodland Ave., Phila., at a cost of 6 cents.

Whey.—This consists of milk from which the casein has been removed by the use of rennet although the lactalbumin remains. The amount of fat is diminished very greatly, being entangled in the curd which the rennet produces. Whey is prepared as follows: Into a quart of warm, fresh milk, heated to 100°F. (37.8°C.) are stirred 2 teaspoonfuls of liquid rennet or of essence of pepsin. After coagulation has taken place the milk is placed in the cold for about $\frac{1}{2}$ hour, the curd then broken up with a fork in order to liberate the whey, and the latter strained through cheese-cloth or muslin without pressure. There will be obtained from 1 to $1\frac{1}{2}$ pints of whey. The finer-meshed and thicker the cloth the more fat will be removed by it. When it is desired to produce a whey containing practically no fat, a fat-free, separator milk or the milk from a quart jar after the cream has been removed should be employed instead of whole-milk. Failure to coagulate firmly indicates that not enough rennet was used or that the milk had been boiled previously or was too cold. If whey is to be mixed later with milk or cream, the rennin remaining in it must be destroyed by heating it for some time to a temperature of 140°F. (60°C.) or over (pasteurizing), otherwise the casein of the cream will coagulate. If it is heated to 75°C. (167°F.) coagulation of the lactalbumin begins. Various analyses of whey have been made. The average of many of them, according to the figures given by König,¹ is as follows:

TABLE 46.—COMPOSITION OF WHEY (KÖNIG)

Fat.....	0.32
Sugar.....	4.83
Whey protein.....	0.85
Salts.....	0.64
Water.....	93.36

It will be seen from this that the percentage of salts is practically unchanged as compared with milk, and that of sugar slightly increased. Certain later analyses differ from these. Thus White and Ladd² found an average of 1.02 per cent. of protein and the Department of Agriculture of the United States³ one of 1.00 per cent.

Whey-cream Mixtures.—Whey was for many years a very favorite food for cases of weak digestion, either given for a brief period alone except for the addition of cane-sugar, or combined with small amounts of cream. Falling for a while into disuse, its employment was revived at the time the fear of the injurious effects of a high casein-percentage became prominent; the purpose being to replace the casein by lactalbumin. In recent years, with increasing confidence in the digestibility of casein, the use of whey has been neglected and even condemned, on the ground that the whey-protein was injurious in many cases of digestive disturbance. This neglect has been harmful, for although the need for whey-combinations is comparatively infrequent, yet there are numerous instances especially in early infancy when they are of undoubted benefit. In fact, the whey is the element which contains the bulk of the necessary amino-acids, the casein being very deficient in these. (See p. 131.) Regarding the harmfulness it is doubtful whether, in any case of injury by whey, this is not to be attributed to the sugar or the salts rather than to the lactalbumin.

The production of whey-cream mixtures in which the fat shall be

¹ *Chemie der menschliche Nahrungs-u. Genussmittel*, 1903, I, 389.

² *Phila. Med. Jour.*, 1901, Feb., 218.

³ *Bulletin No. 28.* Ref. White and Ladd, *loc. cit.*

sufficiently abundant and the casein in very small amount can be accomplished only with the employment of the richer creams. This is because with the weaker creams and top-milks of from 8 to 16 per cent. strength, so much needs to be used to obtain sufficient fat that a very considerable percentage of casein is necessarily added also. The whey-cream mixtures are consequently much better prepared by laboratory-modification when possible. Using, however, the top 2 oz. of the quart, containing 24 per cent. of fat, successful home-modifications can be made. Since the desire usually is to have present as much lactalbumin as possible in the food and as little casein, and since the fat-percentage of the whey is so low and so little cream is employed, we shall make no great error in considering the fat as derived entirely from the cream and the protein and sugar from the whey. Suppose, for instance, that 3 per cent. of fat is desired in a 20 oz. mixture, we can construct a food by using the basic equation previously given (p. 140). Substituting the figures we shall have:

$$\frac{20 \times 3}{24} = 2.5 \text{ oz. of 24 per cent. cream.}$$

The remaining 17.5 oz. will be whey. The total sugar-percentage as derived from the whey equals nearly 5 per cent. (4.83), and as much more sugar may be added as is desired to bring it up to the required percentage. A closer calculation of the whey-formula is hardly necessary. Those, however, who desire to take into account the amount of casein present in the top-milk and of fat in the whey may follow the methods elaborated by Westcott.¹

Peptonized Milk.—For infants with feeble digestion the pancreatizing of the milk before it enters the alimentary canal was formerly much used and was sometimes of benefit. This is best accomplished by the action on the milk of the trypsin derived from the pancreatic juice of the pig, which partially digests the casein before it is ingested and prevents the formation of tough coagula. As the pancreatic extract requires the presence of an alkali in order to act, it is commonly sold combined with bicarbonate of soda, in the form of a powder in individual glass tubes. These are preferable to the "peptogenic" powder on the market, as they permit of greater range in varying the food at will. The pancreatizing is accomplished by dissolving the contents of 1 tube in 2 oz. of cool water and adding to 1 pint of cool milk; and then allowing the vessel containing the mixture to stand in hot water of a temperature of not over 115°F. (46°C.), *i.e.* as warm as the finger can bear without discomfort. It may remain here for 20 or 30 minutes, or a shorter time if the slightest bitter taste is discovered, and is then quickly cooled and kept on ice, or, better, heated quickly to boiling. The temperature of boiling destroys the trypsin and prevents further peptonization, thus avoiding the development of the bitter taste. The peptonizing may be applied similarly to the top-milk, or to the modified milk-mixture after it has been prepared in the ordinary way.

It is not best to continue the use of peptonized milk for too long a time, as it takes away the necessity, and, to an extent, the power of the digestive organs to do their own work. Its employment is serviceable only when there is a disturbance of protein-digestion. It is with reason much less frequently made use of than formerly; but is certainly serviceable in some cases.

¹ Internat. Clinics, 1900, III.

Buttermilk. Lactic Acid Milk.—The term “buttermilk” is properly applied to the milk which remains after the butter-fat has been removed by churning. Its employment for the feeding of infants with delicate digestion, for many years widely followed in Holland, appears to have been first urged upon the profession by Ballot in 1865.¹ Renewed attention was directed to it by the writings of de Jager,² Salge,³ Teixeira de Mattos,⁴ and, later, by many others. The composition of buttermilk varies according to different published analyses, and depends to some extent upon whether made from whole sweet milk, sweet cream, or sour cream. By all methods the fat-percentage is low, that of the protein rather high, and that of the sugar more or less reduced. The following table gives approximate analyses.

TABLE 47.—APPROXIMATE COMPOSITION OF BUTTERMILK

Fat.....	0.5–1.0 per cent.
Sugar.....	0.3–4.0 per cent.
Protein.....	2.5–4.0 per cent.

The average composition from a large number of published statistics collected by König⁵ gives water 90.09, fat 1.02, sugar 4.24, nitrogenous material 3.91, and salts 0.74. Various condensed buttermilks have been put upon the market. These frequently fill a useful place. (See p. 166.) Buttermilk is always distinctly acid from the development in it of lactic acid and contains usually about 0.5 per cent. of this. If the acidity grows too great the buttermilk separates into a whey-like and a more solid portion. The casein is always coagulated and is in the form of casein-lactate and it will no longer be acted upon by the rennin. The caloric value is between 300 and 400 calories per litre (284 and 379 per quart) (see also p. 175), being little if any stronger than skimmed milk. Ordinary commercial buttermilk is hardly fit for use in infant-feeding, as it is liable to be contaminated by undesirable germs. It is best made carefully at home in the following manner: A quart or more of either whole-milk or skimmed-milk, as fresh and clean as possible, is pasteurized and placed in a clean vessel. After the addition of a culture of lactic acid bacilli it is allowed to stand for 18 to 24 hours at a temperature of about 100°F. (37.8°C.). The Bulgarian bacillus cultures are the most serviceable, but other lactic acid germs may be employed. Since there are numbers of cultures on the market, many of which have little value for the purpose (Bendick),⁶ an active variety should be selected. After standing as directed the milk is then churned energetically for 20 to 30 minutes in a small glass churn, the butter removed, and the buttermilk kept on ice until wanted. Instead of churning buttermilk may be made by using skimmed-milk and beating it thoroughly after souring has taken place. Prepared in either way the living lactic acid germs are still present, and are serviceable in some digestive disturbances where it is desired to destroy by their growth the proteolytic bacteria. When only the chemical action of the buttermilk is desired, boiling will destroy the germs. Boiling, however, curdles the casein into large masses, unless vigorous stirring is used to prevent this.

Owing to the low caloric value buttermilk by itself is suitable for

¹ Nederl. Tijdschr. v. Geneesk., 1865, II, 402. Ref. Teixeira de Mattos.

² Nederl. Tijdschr. v. Geneesk., 1895, XXXI, 679.

³ Jahrb. f. Kinderh., 1901, LIV, 681.

⁴ Jahrb. f. Kinderh., 1902, LV, 1.

⁵ Chemie der menschlichen Nahrungs- und Genussmittel, 1903, I, 386.

⁶ Journ. Amer. Med. Assoc., 1915, LXIV, 809.

infant-feeding only for short periods. To increase its strength the usual custom is to add wheat-flour and cane-sugar to it. The amounts employed vary, but the preparation is made in the following manner somewhat after the proportions recommended by Teixeira de Mattos:¹ 1 level tablespoonful ($\frac{1}{4}$ oz.) of wheat, rice or other flour is rubbed into a paste with 6 to 8 oz. of a quart of buttermilk, and $4\frac{1}{2}$ level tablespoonfuls ($2\frac{1}{4}$ oz.) of granulated sugar are added. This is then mixed with the remainder of the quart and the whole boiled for 25 minutes, with constant stirring. If it is desired to preserve the germs alive, the flour and sugar are boiled in the 8 oz. of buttermilk in a double boiler, and then when cool added to the remaining buttermilk. Buttermilk mixtures prepared in this way give approximate percentages of fat 1 per cent., carbohydrate 11 per cent., protein 4 per cent., based on König's figures, but varying somewhat with the buttermilk employed. The advantages of the buttermilk-mixture depend upon the relationship of the percentages. It is serviceable in cases where fat is not at all well tolerated and where there is no intolerance for carbohydrate. The caloric value varies between 560 and 660 calories per quart (592 and 697 calories per litre). It is also useful from the fact that the casein has already been coagulated by the acid, and is in a very finely divided state, not capable of being acted upon by rennin.

Lactic acid milk is a term which might conveniently be limited to whole-milks which have been soured as for buttermilk, but which have not had the butter-fat removed by churning. Koumys and Matzoon are of this class. They are suitable for older children, but cannot, of course, be used where a milk-mixture free from fat is desired.

Casein Milk (Eiweiss Milk. Protein Milk. Albumin Milk).—Although not a translation of the German title "Eiweissmilch" applied by Finkelstein and Meyer,² the title "Casein milk" would appear to express well the composition of the food. It is distinctly a casein preparation, not one of all the proteins of the milk. Its principal purpose is to furnish a diet with a fair amount of fat; a low percentage of sugar, especially lactose; a reduced percentage of salts and of lactalbumin, and a large amount of casein. The percentage-composition³ averages approximately the figures given in the following table:

TABLE 48.—COMPOSITION OF CASEIN MILK

Fat.....	2.5 per cent.
Lactose.....	1.5 per cent.
Protein.....	3.0 per cent.
Salts.....	0.5 per cent.

The amount of the fat is somewhat variable. The caloric value is about 450 per litre (426 per quart). A little saccharin may be used for sweetening if necessary. The formula is based on the belief that the sugar and the salts of the food are the most dangerous elements in many diseased conditions. By diminishing the amount of these the fat is rendered more easily digestible. Later, after the digestion of the infant has improved, the percentage of sugar is raised by the addition of a dextrin-maltose combination.

The preparation of casein milk is rather difficult and requires close attention to details: One quart of whole-milk is heated to about 100°F.

¹ *Loc. cit.*

² Berl. klin. Woch., 1910, XLVII, 1165; Jahrb. f. Kinderh., 1910, LXXI, 525; Münch. med. Woch., 1911, LVIII, 340.

³ Finkelstein and Meyer in Feer's Lehrb. d. Kinderheilk., 1914, 252.

(37.8°C.), $\frac{1}{2}$ oz. of liquid rennet or essence of pepsin added, and the milk then allowed to stand at this temperature in a water-bath for half an hour, by which time it will have been curdled. The mass is then put upon a fine cloth and the whey strained off without pressing, about an hour being allowed for this. The curd is then rubbed through a very fine wire sieve, using the bowl of a spoon to do the rubbing, and a pint of water being used in the process; and this sieving repeated 4 or 5 times, using the same water and later adding if necessary enough to preserve the original volume. To the pint thus obtained 1 pint of buttermilk is added. The whole is then sterilized by boiling vigorously and constantly stirring during the process in order to prevent clumping.

Owing to the difficulty in preparation, or as a result of efforts made at improvement, many substitutes have been recommended and some of them produced commercially and put upon the market (see p. 168); but a very similar preparation may be easily made at home by the method proposed by Hoobler.¹ This consists in mixing 10 grams (154 grains) (about $1\frac{1}{3}$ level tablespoonfuls) of powdered casein (casein-flour) with 1 pint of previously boiled fat-free buttermilk and 1 pint of warm water. This gives a percentage composition of approximately fat 0.25 per cent., carbohydrate 2 per cent., protein 2.8 per cent. The formula can be varied in different ways according to the needs of the infant. The following formulæ serve as examples of those employed in the Children's Hospital of Philadelphia. In some of them a portion of the milk used is fat-free buttermilk and a part lactic acid milk. (See p. 148.) Larosan (p. 168) may be employed instead of casein-flour if desired, and the mixture resulting is finer and can be boiled without thickening.

TABLE 49.—COMPOSITION OF VARIOUS CASEIN-MILK FORMULÆ

Buttermilk, ounces	Lactic acid milk, ounces	Casein-flour	Dextrin-maltose	Water, ounces
(1) 10	0	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	10
(2) 5	5	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	10
(3)	10	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	10
(4)	15	6 grams (about 1 scant level tablespoonful)	18 grams (about 3 scant level tablespoonfuls)	5

These give the following approximate calculated percentages and caloric value:

	Fat	Carbohydrate	Protein	Calories per ounce
(1)	0.15	5.25	2.75	10.4
(2)	1.10	5.25	2.75	12.7
(3)	2.00	5.25	2.75	15.0
(4)	3.00	6.40	3.60	20.0

These calculations are based upon the same approximate percentage-strengths employed in using ordinary home-modifications from top-milk (p. 139), and are open to the same criticism of lack of absolute accuracy. The variation is, however, insignificant.

¹ Arch. of Ped., 1914, XXXI, 174.

CHAPTER V

FOODS OTHER THAN MILK

PROTEIN-FOODS

It happens constantly that some animal substance other than milk is needed as a temporary substitute during illness in the 1st year; or as a more permanent article of diet after this period. The following may be mentioned:

Albumen-water.—This consists of the white of one hen's egg, averaging from 55 to 60 grams (Pfund)¹ (1.9 to 2 fl.oz.) stirred in enough cool water to make 8 oz., and strained if necessary. It is an excellent temporary substitute for milk in acute cases of failure of digestion, but a very weak one. It is also at times a valuable addition to cream-and-water mixtures, or cream-and-whey mixtures, when it is desired to increase the protein of the food by the use of some substance other than casein. The possibility of it being directly absorbed as a foreign albumin when intestinal disturbance is present is not to be forgotten. A sensitization to egg might be produced in this way. According to Hammarsten² white of egg contains 10 to 13 per cent. of protein, a trace of fat, 0.7 per cent. of salts, and 85 to 88 per cent. of water. When mixed with enough water to make 8 oz. a protein-percentage of about 1.3 is obtained. A certain proportion of the egg-white, however, appears in the water in the form of shreddy material, and requires to be removed by straining. The actual protein-percentage may thus be reduced considerably. Albumen-water may be given cool or slightly warm, with or without sugar or with a pinch of salt, and from a glass or bottle according to the taste and fancy of the infant. It may be flavored with a few drops of lemon or orange juice or with whiskey. According to Stutzer³ 1 lb. of lean beef equals 15 eggs in proteid constituent.

Fresh Beef-juice.—This may best be prepared in either of two methods:

1. Season with salt and very slightly broil a piece of steak free from fat, then cut it into small pieces and express the juice with a meat press (Fig. 25). A lemon squeezer may be used instead, but this method is more wasteful. A pound of beef will make about 2 or 3 fl.oz. of juice. The juice must be kept on ice until needed. It may then be warmed slightly or given cold with a spoon. Some infants prefer it with the addition of sugar.

2. A pound of minced steak is put into 6 or 8 oz. of water and salted slightly. This is allowed to stand on ice over night. It is then squeezed out well with the meat press or is strained through muslin by twisting it tightly. Juice thus obtained is somewhat thinner than by the first method, but is decidedly larger in quantity.

The protein of beef-juice varies in amount, depending upon the method employed in obtaining it. According to the statistics given by Hutchi-

¹ Zeitsch. f. Hyg., 1900, XXXV, 444.

² Phys. Chemic, 1904, 435.

³ Centr. f. allg. Gesundheitspflege, 1882, I, 179.

son¹ it varies from 2 to 7 per cent. Its fat-percentage is low. Beef-juice is of value as a condensed, stimulating food for infants who have difficulty in digesting the protein of milk, but its caloric value is low and it is not sufficient for an exclusive diet in the quantity which the digestion will safely tolerate. Its administration may be alternated with albumen-water or cereal decoctions in cases where it is desired to avoid milk for a time. The daily amount should be 1 to 2 teaspoonfuls at first and gradually increased to 3 or 4 oz. Beef-juice is also useful in cases of anemia or deficient nutrition as an adjuvant to the milk-diet, and may then be added in quantities of 2 or 3 teaspoonfuls to each bottle, or, preferably, given separately in 1 or 2 portions in the twenty-four hours between the regular feedings. In the 2d year of life beef-juice may well constitute a part of the daily food for healthy children.

Minced Rare Beef.—A thick piece of underdone steak, as free from fat and tendon as possible, is scraped, grated, or minced very fine; pounded in a mortar until it is reduced to a pulp, and seasoned with salt. For infants it may now be rubbed up with a little water until it is of the consistency of thick cream, and fed from a spoon, giving 3 or 4 teaspoonfuls in the course of the day.

Scraped beef is a very concentrated form of nitrogenous food, sometimes useful even in subjects less than 1 year of age. In the 2d year 1 or 2 tablespoonfuls of the scraped meat without water may be given daily when casein cannot be digested. I have more than once seen tape-worm develop from the use of raw beef given in this way, but the danger may be avoided entirely by the slight broiling referred to.

Meat Broths.—Foods of this class are of practically no value as actual nourishment, if of the class of "clear broth." They are sometimes of benefit during the 1st year as a temporary substitute for milk-mixtures, and in the 2d year may be one of the ordinary articles of diet. The principles and method of preparation are the same for all, and the following description for making beef-broth applies to the others: 1 pound of lean meat is cut into small pieces, and these, together with portions of the cracked bones put into 1 pt. of cold water. This is heated very slowly, allowing it to simmer for 3 or 4 hours; replacing the water as it evaporates; and then strained, cooled and the fat removed. The refraining from straining, thus leaving in the finely divided muscle-fibre, increases the nutritive value very decidedly. The addition of a cereal flour also adds to the value as a food, and makes it a very serviceable article of diet in the 2d year, or for temporary use to replace milk in digestive disturbances in the 1st year.

Beef-tea.—This may be prepared as follows: (1) Scrape or cut fine 1 pound of lean meat and place in 1 pint of cold water in a jar. Let it stand in the cold for 1 hour, stirring occasionally. Then heat the jar in a saucepan of water at not over 167°F. (75°C.) for another hour, stirring occasionally. It should then be raised to the boiling point for a moment, strained, cooled and the fat removed. Warm slightly and season before giving it. In place of straining, the liquid may be simply poured off.

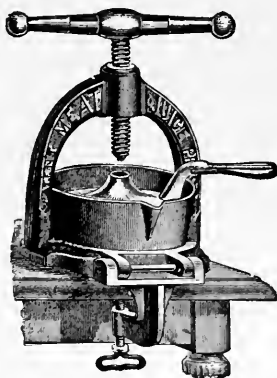


FIG. 25.—MEAT PRESS.

¹ Food and Dietetics, 1901, 97.

To this should be added the additional liquid obtained by firmly pressing the pieces of meat which remain. (2) Another more rapid method is the following: Place the pound of meat in $\frac{1}{2}$ pint of boiling water. Keep this gently warm for 10 minutes; strain; cool rapidly and remove the fat.

Beef-tea is distinguished from beef-broth particularly by the considerable amount of gelatin contained in the latter. This is derived from the prolonged boiling of the bones and of the connective tissue. Beef-tea has little nutritive value, and practically none at all if sufficient heat has been used to coagulate the protein, and if this has been removed by straining. If the fluid is not strained, the food-value is decidedly increased.

Veal Tea.—This preparation, formerly much in vogue, is very similar in its composition and in its strength to beef-tea. It may be made as follows: Cut fine 1 pound of veal as free from fat as possible, put into $1\frac{1}{2}$ pints of cool water; let stand in the cold for 1 hour; then keep warm without boiling for 3 or 4 hours; strain; let cool and skim off the fat.

Gelatin.—About 20 grains of gelatin are soaked for a short time in cold water, and then dissolved with stirring in $\frac{1}{2}$ pint of boiling water. Gelatin as an element of milk-mixtures was very popular in former years. Its mechanical action in the food is very similar to that of barley-water. Chemically it possesses but little food-value.

Soy Bean Flour.—This substance may be properly classed among protein-foods serviceable for temporary use when, for any reason, the protein of milk is not desired. According to Ruhräh¹ it contains approximately 35 to 40 per cent. of protein, 4 per cent. of fibre, 9 per cent. of starch, sugar and dextrin, and 18 to 19 per cent. of fat. Made into a flour the protein is over 44 per cent.

AMYLACEOUS FOODS

The employment of starchy foods in the 1st year has already repeatedly been referred to. (See pp. 124, 130, 141, 144, etc.). Although the power of digesting starch is present in early infancy, there is no need for the administration of it to the healthy normal artificially fed infant during the early part of the 1st year. The addition of starch to the diet in the latter part of the year is usually advantageous in order to accustom the child to this food. In the case of illness, however, weak or stronger amylaceous additions or substitutions are often of the greatest value, and this is particularly true when there is difficulty in the digestion of sugar. Starch is also used to prevent the casein from forming tough coagula, approximately 0.7 per cent. of starch in the milk-mixture being the best amount to accomplish this (White).² For infants in the 1st year the amylaceous food is generally either a cereal-flour or arrowroot, and is usually given in the form of a decoction of various strengths. After this period cereal-gruels become a recognized part of the diet.

The Weight of Various Carbohydrate Substances. (See also p. 135).—In the making of amylaceous decoctions in the home, measuring by bulk is usually the only method which can be employed; but for better knowledge it is necessary to know the weight also, in order to estimate the percentages obtained. Tablespoons are the measure oftenest used, but as these vary so much in size, it is better to employ the Chapin dipper or other ounce measure. I have made a series of weighings,

¹ Arch. of Ped., 1909, XXVI, 496; Amer. Jour. Med. Sci., 1915, CL, 502.

² Journ. of Bost. Soc. of Med. Imp., 1900, V, 125

with the results shown in the table which follows. They correspond fairly closely with those determined by Chapin¹ and with those obtained for me by Dr. A. Græme Mitchell. The figures can be only average ones, especially as regards the weight of the tablespoonful. Barley and oat flours are slightly lighter, and rice and wheat flours slightly heavier than the figures given. In measuring, the tablespoon or the dipper should be filled from a smaller spoon, tapped sharply 2 or 3 times, and the excess scraped off with a case-knife. Too much tapping settles the contents too greatly. Casein-flour is included as a convenience.

TABLE 50.—APPROXIMATE WEIGHTS OF GRAINS, FLOURS AND SUGARS
(Measured by Tablespoon and by Fl. oz. Dipper)

1 Level tablespoonful of barley or other flour weighs about $\frac{1}{4}$ oz. Av.
1 Level dipper of barley or other flour weighs about $\frac{1}{2}$ oz. Av.
1 Level tablespoonful of Bermuda arrowroot weighs about $\frac{2}{5}$ oz. Av.
1 Level dipper of Bermuda arrowroot weighs about $\frac{4}{5}$ oz. Av.
1 Level tablespoonful of pearl barley weighs about $\frac{1}{2}$ oz. Av.
1 Level dipper of pearl barley weighs about $\frac{3}{4}$ oz. Av.
1 Level tablespoonful of rice weighs about $\frac{1}{2}$ oz. Av.
1 Level dipper of rice weighs about $\frac{4}{5}$ oz. Av.
1 Level tablespoonful of rolled oats weighs about $\frac{1}{6}$ oz. Av.
1 Level dipper of rolled oats weighs about $\frac{1}{3}$ oz. Av.
1 Level tablespoonful of lactose weighs about $\frac{1}{3}$ oz. Av.
1 Level dipper of lactose weighs about $\frac{3}{4}$ oz. Av.
1 Level tablespoonful of dextrin-maltose weighs about $\frac{1}{4}$ oz. Av.
1 Level dipper of dextrin-maltose weighs about $\frac{1}{2}$ oz. Av.
1 Level tablespoonful of saccharose weighs about $\frac{1}{2}$ oz. Av.
1 Level dipper of saccharose weighs about 1 oz. Av.
1 Level tablespoonful of casein-flour weighs about $\frac{1}{4}$ oz. Av.
1 Level dipper full of casein-flour weighs about $\frac{1}{2}$ oz. Av.

Strength of Cereal Gruels.—It is of distinct advantage to know with approximate accuracy the actual amount of the ingredients contained in the various cereal gruels employed. The matter has been studied by Chapin² and others. The following Table gives the approximate percentage formula of some of these gruels.³ (Table 51.)

The fat and mineral matter are present in such small amounts that they may be ignored for practical purposes, and it is hardly necessary to take the protein into consideration. Simple gruels cannot be made stronger than about 2 oz. of the flour to the quart. If more concentrated preparations are desired they must be dextrinized (p. 155).

Various Starchy Decoctions. **Barley-water.**—Barley-water is made either from pearl barley or from barley-flour. With the former 2 level tablespoonfuls of pearl barley, previously well washed for some time in cold water, are placed in 1 quart of water, let simmer slowly until the liquid is reduced to 1 pint in volume, and then strained. This should be quite fluid in consistency when slightly warmed. In place of the whole grain 1 level tablespoonful of barley-flour may be boiled slowly in 1 pint of water for 15 minutes, with stirring, and then strained, replacing the water which has boiled away. This has the advantage that it can be made much more rapidly and conveniently. As with all amylaceous decoctions it should be prepared fresh daily and kept on ice, as it sours readily.

Barley-water is a very commonly used temporary substitute for milk

¹ Med. Rec., 1905, LXVII, 246.

² Loc. cit.

³ The analyses are those published by the Cerec Company of Tappan, New York, and based on gruels made of flours furnished by them.

TABLE 51.—PERCENTAGE COMPOSITION OF GRUEL

	Barley		Legume		Oat		Wheat	
	Proteids, per cent.	Carbo- hydrates, per cent.	Proteids, per cent.	Carbo- hydrates, per cent.	Proteids, per cent.	Carbo- hydrates, per cent.	Proteids, per cent.	Carbo- hydrates, per cent.
1 Level tablespoonful flour ($\frac{1}{4}$ oz. Av.) to quart of gruel.....	0.12	0.60	0.19	0.53	0.12	0.60	0.10	0.62
2 Level tablespoonfuls flour ($\frac{1}{2}$ oz. Av.) to quart of gruel.....	0.24	1.20	0.39	1.06	0.24	1.20	0.20	1.25
3 Level tablespoonfuls flour ($\frac{3}{4}$ oz. Av.) to quart of gruel.....	0.36	1.80	0.58	1.59	0.36	1.80	0.30	1.88
4 Level tablespoonfuls flour (1 oz. Av.) to quart of gruel.....	0.48	2.40	0.78	2.12	0.48	2.40	0.40	2.50
8 Level tablespoonfuls flour (2 oz. Av.) to quart of gruel.....	0.96	4.80	1.56	4.24	0.96	4.80	0.80	5.00
12 Level tablespoonfuls flour (3 oz. Av.) to quart of gruel.....	1.44	7.20	2.34	6.36	1.44	7.20	1.20	7.50
16 Level tablespoonfuls flour (4 oz. Av.) to quart of gruel.....	1.92	9.60	3.12	8.48	1.92	9.60	1.60	10.00
20 Level tablespoonfuls flour (5 oz. Av.) to quart of gruel.....	2.40	12.00	3.90	10.60	2.40	12.00	2.00	12.50
24 Level tablespoonfuls flour (6 oz. Av.) to quart of gruel.....	2.88	14.40	4.68	12.72	2.88	14.40	2.40	15.00
28 Level tablespoonfuls flour (7 oz. Av.) to quart of gruel.....	3.36	16.80	5.46	14.84	3.36	16.80	2.80	17.50
32 Level tablespoonfuls flour (8 oz. Av.) to quart of gruel.....	3.84	19.20	6.24	16.96	3.84	19.20	3.20	20.00

in cases of acute indigestion where abstinence from food is the best cure. Its nutritive power being extremely slight, it is given in this case not so much for nourishment as for the allaying of hunger at the time. When used as a diluent of milk-mixtures it is supposed to have a somewhat constipating action, but this is by no means always the case.

Barley-jelly is the term applied to a thicker barley-decoction which jellies firmly when cool. It is made by using 4 level tablespoonfuls of pearl barley to the quart of water, or 3 of barley-flour to the pint. A double boiler may be used to advantage to prevent burning.

Oatmeal-water.—This may be made in the following manner: Put 2 level tablespoonfuls of oatmeal into 1 pint of water. Let simmer for 2 hours or more, replacing the water as it evaporates; strain. An *oatmeal-jelly* may be made in a way similar to that employed for making barley-jelly, using 4 level tablespoonfuls to the pint of water, and cooking as for oatmeal-water, although in a double boiler. Oatmeal-water or oatmeal-jelly is used with milk as are the analogous preparations of barley. It is, however, distinctly laxative to many children, and is often a useful addition where there is constipation. In many cases oatmeal disagrees decidedly. If amylaceous food is to be added during the 1st year, some other than oatmeal should generally be first tried.

Rice-water.—The method employed is the adding of 2 level tablespoonfuls of washed rice to 1 quart of water, boiling to 1 pint and straining, and then mixing with the food in the same proportions as with barley-water. It is supposed to have a constipating effect, and is often used when there is a tendency to diarrhea. Its actual value for such a purpose is doubtful. In place of the rice-grain, rice-flour may be employed, using 1 level tablespoonful to 1 pint of water, boiling for 15 minutes and straining. Rice is richer in carbohydrate and poorer in protein than the other cereals mentioned.

Arrowroot.—This is a serviceable food when a starchy addition is required. It is almost a pure starch, containing almost no protein. It may be given in the form of *arrowroot-water*, which is made by adding 1½ level tablespoonfuls of Bermuda arrowroot to 1 pint of water and boiling for 5 or 10 minutes. *Arrowroot-jelly* is made by using 4 level tablespoonfuls of arrowroot to the pint of water, and boiling for a few minutes in a double boiler.

Bean-flour.—This has been employed in the form of a gruel when, with the addition of carbohydrate, it is desired to increase the protein-content of the food decidedly by the use of a vegetable product rich in this substance. The Soy bean (p. 152), recommended for infant-feeding especially by Ruhräh,¹ contains so little starch that it cannot be properly classified among amylaceous foods.

Dextrinized (MalTED) Starch.—The addition of malt-extract to boiled starch rapidly dissolves it, transforming it into dextrin and maltose and intermediate substances. Starch, as such, ceases to be present if the dextrinizing is complete. Treated in this way it becomes very absorbable and, added to the milk-mixture, often aids greatly in nourishing infants with feeble powers of digestion or assimilation of fat or of lactose. The various commercial dextrin-maltose preparations on the market are of this class. (See pp. 129, 163.) As made at home, 1 fluidrachm of a powerful diastatic malt extract, such as Cerec, will transform the starch of 10 oz. of a 10 per cent. amylaceous decoction. According to investigations made for me by Dr. A. Graeme Mitchell at the Children's

¹ Arch. of Ped., 1909, XXVI, 496; Amer. Jour. Med. Sci., 1915, CL, 502.

Hospital of Philadelphia, $\frac{1}{3}$ of the starch of such a decoction of arrow-root was converted into a copper-reducing substance in 15 minutes at a temperature of 140°F. (60°C.), and a longer time or a larger amount of the extract would convert all or the greater portion of it. 7 grains of potassium carbonate should then be added to each ounce of the mixture. The dextrinized preparation may replace in part the water of the milk-mixture. The malt-extract should be added to the cereal-jelly while the latter is still warm, about 120° to 150°F. (48.9 to 65.5°C.) and maintained at this temperature, with occasional stirring, for 20 to 30 minutes. Dextrinized starch produced in this way, as also with the commercial dextrin-maltose preparations, has an action different from that of the pure sugars. These latter become very promptly assimilable, while the dextrin of the dextrin-maltose possesses a colloidal action resembling that of starch, and is also slower in being transformed and consequently in being absorbed. The relative amount of dextrin and maltose present depends upon the temperature employed. (See p. 129.) If this is below 55°C. (131°F.), there is more maltose resulting; if above 63°C. (145.4°F.), more dextrin. If above 75°C. (167°F.) the diastatic ferment is destroyed (Morse and Talbot).¹ The strength of milk-mixtures is very considerably increased in the percentage of carbohydrate when dextrinized gruels are employed. The degree of increase can be approximately determined by estimating the amount of amylaceous flour employed (see pp. 153, 154), ignoring the content of the malt-extract if but little of this has been used.

Malt-soup.—A sharp distinction is to be drawn between the food in which malt-soup takes a part, and that to which simple dextrin-maltose preparations or completely dextrinized gruel has been added. With the latter no starch is present, while with malt-soup there is a very decided proportion of this intentionally remaining unconverted. The dextrinized preparation made at home as just described is a malt-soup if the process is not continued to the complete converting of the starch. The preparation was first made and urged by Liebig,² but was later modified by Keller,³ and came rapidly into prominence as a serviceable form of nourishment for a certain class of sick infants. Its value rests on the fact that a large percentage of unconverted starch, together with maltose and dextrin, is present in the food. The method of preparation as commonly advised is as follows:

1. $1\frac{3}{4}$ oz. Av. ($6\frac{1}{2}$ level tablespoonfuls) of wheat-flour are mixed with 11 fl. oz. of cold cow's milk and rubbed through a sieve.

2. $2\frac{1}{2}$ fl. oz. of malt-soup extract are added to 22 fl. oz. of warm water.

The two are then added one to the other and the whole heated slowly to boiling, with constant stirring, and enough water finally added to replace that which has evaporated. The malt-soup-extract should contain 7 grains of carbonate of potash to the fluidounce, the object of this being to alkalinize it and thus to remove its diastatic action, as also to prevent the development of any acidosis in the infant. The caloric value of this mixture is about 625 calories per quart (660 per litre). Some of the malt-extracts upon the market already have the alkali added;⁴ others must have this added before using.⁵ The formula given produces

¹ Diseases of Nutrition and Infant Feeding, 1915, 195.

² Suppe für Säuglinge, 1865.

³ Malzsuppe, eine Nahrung für magendarmkranke Säuglinge, 1898.

⁴ Maltine Co.; Loefflund; Borchardt.

⁵ Freilhofer Co.; Neutral Maltose.

percentages of approximately fat 1.33, carbohydrate 11.4, protein 1.58. It is not, however, necessary to use these amounts. Experiments conducted for me at the Children's Hospital of Philadelphia by Dr. A. Graeme Mitchell evolved the following formulæ which will be found convenient.

TABLE 52.—PERCENTAGE-STRENGTHS VARIOUS MALT-SOUP MIXTURES

TABLE 57. PERCENTAGE STRENGTHS OF FOODS THAT BOIL IN WATER		Per cent.
(a)		
Wheat-flour, $\frac{1}{2}$ oz. (2 level tablespoonfuls)	}	Fat = 0.35
Skimmed milk, 10 oz.		Carbohydrate = 7.5
Malt-soup-extract, 1 oz.		Protein = 2.24
Water to make 20 oz.		
(b)		
Wheat-flour, $\frac{1}{2}$ oz. (2 level tablespoonfuls)	}	Fat = 1.0
Whole-milk, 5 oz.		Carbohydrate = 7.5
Skimmed milk, 5 oz.		Protein = 2.24
Malt-soup-extract, 1 oz.		
Water to make 20 oz.		
(c)		
Wheat-flour, $\frac{1}{2}$ oz. (2 level tablespoonfuls)	}	Fat = 2.0
Milk, 10 oz.		Carbohydrate = 7.5
Malt-soup-extract, 1 oz.		Protein = 2.24
Water to make 20 oz.		
(d)		
Wheat-flour, 1 oz. (4 level tablespoonfuls)	}	Fat = 2.0
Milk, 10 oz.		Carbohydrate = 11.0
Malt-soup-extract $1\frac{1}{2}$ oz.		Protein = 2.6
Water to make 20 oz.		
(e)		
Wheat-flour, $1\frac{1}{2}$ oz. (6 level tablespoonfuls)	}	Fat = 2.1
Milk, 10 oz.		Carbohydrate = 13.84
Malt-soup-extract, $1\frac{3}{4}$ oz.		Protein = 2.86
Water to make 20 oz.		

In these calculations the same approximate percentages were employed as adopted for the making of ordinary home-modifications from top-milk. (See p. 139.)

Flour-ball.—Flour-ball was formerly much employed as an addition to milk when a cereal was required. It is made by tying a pound of flour tightly in a bag and boiling for 10 hours. When cold it is taken from the bag and completely dried with heat. The outer coating is then removed, and the remaining inner portion grated. 1 or 2 teaspoonfuls are added to each bottle. The advantage claimed for it was that the flour was partially dextrinized and thus rendered more soluble, requiring less cooking. Dextrinized cereal flour can, however, be produced much more easily in the manner already described.

CHAPTER VI

SPECIAL NAMED MIXTURES AND PROPRIETARY FOODS

SPECIAL MIXTURES WITH PERSONAL NAMES

There have been recommended a very large number of special mixtures to which are often attached the names of the physicians first describing them. A few of these are mentioned below on account of the frequency with which the names are met with, not because of a desire especially to advocate them. It must be remembered that modern scientific pediatrics is opposed to the use of any one preparation as a routine feeding. Rather must the mixture be modified to meet the needs of the case. Some of the preparations mentioned have indeed been manufactured and sold commercially, and would be more properly included in the next section.

Meigs Gelatin Food.—This was a formula found very useful years ago by J. F. Meigs of Philadelphia, and mentioned here because the title is still widely known. It consisted of 1 scruple of gelatin dissolved in 8 oz. of boiled water to which was then added 1 teaspoonful of arrowroot with milk and cream in varying proportions, depending upon the age and digestive power of the child (Meigs and Pepper).¹

Biedert's Cream Mixtures (Rahmgemenge).²—These consist of modified milks constructed by mixing certain proportions of cream with milk, water, and milk-sugar, thus producing a series of formulæ. The principle is thoroughly scientific and the food was the prototype of many milk modifications later made in Germany, as well as of all percentage feeding combinations. The chief objection to the employment of these mixtures is that, according to the formulæ and the percentages given, the altering of the percentage of one ingredient without affecting that of another is not easy. Later a condensed modified cream-mixture³ prepared according to Biedert's formula, was placed on the market. It will be found mentioned under Commercial Foods (p. 166).

Gärtner's Mother Milk (Fatty Milk).⁴—This is made by centrifugating diluted milk and adding to the richer portion milk-sugar and an alkali. The mixture is then sterilized. The food is in reality a modified milk. It is supposed to possess the same percentages of fat, sugar and protein as are contained in average human milk. It is subject to the disadvantage of all such mixtures, that the relationships of the ingredients to each other are fixed. As the food has become a commercial one its analysis will be given later (p. 166).

Von Dungern's Renneted Milk.⁵—The milk is treated with rennet and the coagulated mass finely divided by shaking. It is claimed that it is rendered more digestible in this way, since the formation of large curded masses in the stomach is prevented. The rennet used is contained in a preparation with the trade name of Peginin.

¹ Diseases of Children, 1877, p. 332.

² Beidert, Der Kinderernährung im Säuglingsalter, 1900, 189.

³ Künstliche Rahmgemenge; Ramogen.

⁴ Wien. med. Wochenschr., 1894, XLIV, 1870.

⁵ Münch. med. Wochenschr., 1900, No. 48.

Szekely's Casein-free Milk.¹—Skimmed milk is heated at about 60°C. (140°F.) in closed vessels under pressure and subjected to liquid carbonic acid which largely precipitates the casein. The whey remaining is mixed with cream and sugar.

Voltmer=Lahrmann's Pancreatized Milk.²—This is merely a pancreatized mixture of cream, milk, water and sugar, with potassium carbonate in varying amounts, to which later phosphoric acid is added.

Backhaus' Milk.³—This consists of fat-free milk treated with rennet. In this way a portion of the casein is coagulated and then removed. To the whey remaining milk-sugar and cream are then added. More recently maltose and dextrin are used. The food has now become a commercial one, three different strengths being sold (p. 166). The preparations are very similar in percentages to those obtained by Biedert's formulæ.

Monti's Whey Milk.⁴—The food consists in varying mixtures of whey and whole milk rich in fat. In this way the lactalbumin is increased in amount and the casein relatively diminished.

Steffen's Veal Broth and Milk⁵ is a mixture of 100 grams (3.4 fl.oz.) each of veal-broth and milk to which are added 1 teaspoonful of cream and 3.8 grams (0.13 fl.oz.) of milk-sugar.

Hesse-Pfund's Infant's Food.⁶—Water and cream are mixed in certain proportions and to them is added a sterilized powder, consisting of hen's eggs and milk-sugar with a small amount of a salt of iron. The amounts of the non-coagulable protein, fat, and sugar are thus increased.

Vigier's Humanized Milk.⁷—The method of preparation consists in first dividing a quantity of milk into two equal portions. On the first half the cream was allowed to rise and was removed, and from the fat-free remainder whey was prepared. This and the cream were then added to the second half of the milk. The resulting mixture was to some extent like that of some of Biedert's cream mixtures, but the percentages were inferior.

Lehndorf and Zak Dialized Milk.⁸—This preparation was especially recommended for cases where there was indigestion of sugar. It is made by placing about a pint of milk in a parchment bag and suspending this in a number of quarts of water, renewing every hour. The sugar and salts pass to a large extent through the parchment.

Homogenized Milk. (Raudnitz).⁹—The milk is minutely divided by atomizing under high pressure. This renders the fat-globules exceedingly small. The cream will no longer rise and the coagulum with acid resembles that of human milk.

Feer's Milk Preparation.¹⁰—This is composed of whole milk 500 grams (16.9 fl.oz.), cream (20 per cent.) 50 grams (1.7 fl.oz.), Soxhlet's Nährzucker 10 to 50 grams (0.35 to 1.8 oz. Av.), plasmon 15 grams (0.53 oz. Av.), water 600 grams (20.29 fl.oz.). This gives a preparation

¹ Münch. med. Wochenschr., 1905, LV, 878.

² Ref. Raudnitz, in Pfaunder u. Schlossmann, Handb. der Kinderh., 1906, I, 311.

³ Berl. klin. Wochenschr., 1895, XXXII, 561; 589. Berl. Molkereiztg., 1905, No. 42.

⁴ Ref. Raudnitz, Monatsschr. f. Kinderh., 1905, IV, 583.

⁵ Kinderheilkunde, 1899, I, 158.

⁶ Jahrb. f. Kinderheilk., 1895, XL, 421.

⁷ Archiv für Kinderheilk., 1898, XXIV, 226; 1903, XXXVI, 407.

⁸ Société de therap., 1893, Jan. 25. Ref. Marfan, Traité de l'allaitement, 1903,

439.

⁹ Wien. med. Wochenschr., 1910, LX, 1930.

¹⁰ Pfaunder u. Schlossmann, Handb. d. Kinderh., 1906, I, 1, 310.

¹¹ Jahrb. f. Kinderh., 1913, LXXVIII, 1.

consisting of fat 2.3 per cent., sugar 6.2 per cent., protein 2.6 per cent., salts 0.44 per cent. The purpose of the food was to render the fat better tolerated by the removal of part of the whey. The lack of protein caused by the dilution is made up by the plasmon.

Schloss' Modified Milk.¹—This is a cream, whole milk and water mixture to which Soxhlet's Nährzucker, mondammin, and nutrose or plasmon have been added.

Friedenthal's Milk.²—The principle of the Friedenthal milk is based especially on an effort to render the mineral matter of the food similar to that of human milk. An artificial human-milk serum is made by dissolving in water the required salts. To this white of egg and powdered casein are added, and the mixture used as the basis for the infant's food.

PROPRIETARY (COMMERCIAL; PATENTED) FOODS

The so-common and wide-spread employment of the proprietary infant-foods is probably one of the most pernicious factors of the time in the feeding of infants, as indeed it has been for years. It is not because the composition of these foods is necessarily faulty which renders them so harmful, although this is frequently the case, but it is the manner in which they are freely advertised to the laity as "the only proper substitute for mother's milk," and freely used by mothers without consultation with physicians. It is frequently only after an infant becomes ill, having taken a large variety of the foods for weeks or months, that the advice of the physician is asked about the propriety of their use. In other cases physicians themselves are largely responsible for this state of affairs, as they are prone to yield too readily to the temptation to prescribe the foods without due consideration. This saves the physician thought, and is the worst thing possible both for him and for the patient.

The proprietary foods are unreliable and unnecessary:—unreliable, because they are never the perfect substitute for mother's milk in spite of the claims of the manufacturers; unnecessary because it is rare that they cannot be entirely dispensed with. Moreover, in the case of those intended to be used without the addition of milk, the element of freshness, so important in infant-nourishment, is lacking; while in the case of the others the question arises, why use them at all if fresh cow's milk must also be employed in any event? They also have the additional disadvantage that the ingredients bear a fixed, unvarying relationship to each other, and the ready changing of these, which modern infant-feeding considers indispensable, is an impossibility. It is true that many infants have done well upon commercial foods after some milk-modifications had failed; but they would almost certainly have grown as satisfactorily without them and with much less risk. What they needed was a proper milk-modification. There can be nothing of advantage in a proprietary food which cannot be equally incorporated in a home-made modification, and at much less expense. It is still more true that not only have countless deaths arisen from their use, but that countless infants have suffered from rickets, scurvy, and severe gastro-intestinal disorders as a result of their employment. Cases do occasionally occur where the temporary use of a proprietary food may be a necessity because nothing else can be obtained or for some other unusual reason; or beneficial, just as barley-water, beef-juice, or other food than milk is sometimes demanded. These

¹ Ueber Säuglingsernährung, 1912.

² Zentralbl. f. Physiol., 1910, XXIV, 687.

cases are certainly the exception; and it is most important that when the selection of a proprietary food under such circumstances is to be made, the physician should be fully cognizant of its composition and what may be expected from it.

There are a number of preparations on the market consisting almost entirely of starch, or of some form of sugar, or of protein, and claiming nothing else, which may with propriety be used whenever the addition of one of these substances to a milk-mixture is to be employed; and if these are not advertised to the laity, and consequently liable to be given by the mother without professional advice, no objection can be raised to them.

The number of proprietary foods which are now or have been on the market is vast; and it would be a waste of time and space to attempt to consider them all. Only a comparatively few will be referred to, in order that the reader may have some idea of their actual composition and mode of manufacture. The analytical tables are given in order to show how closely, or oftener how remotely, they resemble human milk in composition. Some of the foods are still actively employed; of others little is now heard.

The various proprietary foods may be classified as follows:

I. *Condensed Milks*.—These consist of whole or skimmed milk condensed by evaporating, and often with the addition of cane-sugar.

II. *Malted or Dextrinized Foods*.—The basis of these is starch which has been completely converted. To some of them milk has been added in the process of manufacture. A few have still other additions.

III. *Amylaceous Foods*.—These are composed entirely or partly of unconverted starch. Quite commonly it is recommended that they be mixed with milk. Milk and milk-sugar have been added in the process of manufacture of some of them.

IV. *Miscellaneous Foods*.—Infant-foods often with milk as a basis with the addition of other substances than, or in addition to, starch or sugar. They are incompletely or not at all dextrinized.

V. *Protein Foods*.—These consist of commercial foods claimed to be especially rich in protein. They are not primarily intended for use in infancy, but are often of temporary benefit.

1. *Condensed Milks*.—These, including the so-called “dried milks” and “evaporated creams,” are made by evaporating the milk by heat to a greater or less degree and then sealing it in cans. All of them need much dilution with water before they can be employed.

They may be divided into:

1. *Dried Milk*.—This is, in fact, a condensed milk, but not in the usual commercial sense of the term. None of the condensed milks, as ordinarily designated, have been condensed to the degree of actual dryness. There are various preparations of dried milk upon the market sold under different names. Some of them are made from whole milk; others from skimmed milk. The analysis given on p. 162 readily shows to which of these two classes each belong. Mammala is produced from skimmed milk, with the addition of lactose.

2. *Unsweetened and Condensed Whole Milk*.—In this form nothing has been removed by skimming and nothing added. It is simply a condensed whole milk. It keeps badly after the receptacle is opened.

3. *Sweetened and Condensed Whole Milk*.—In this instance from 40 to 45 per cent. of saccharose has been added in the course of preparation for the purpose of preserving the milk.

4. Sweetened and Condensed Skimmed Milk.—Here the milk has had the fat removed, and cane-sugar added as in the previous form.

All the condensed milks are unsuitable for infant-feeding. When properly diluted with water the ingredients still retain at the best the normal relationships of cow's milk. They are all deficient in the amount of fat required by the normal infant after the protein-percentage has been properly reduced. They may be used temporarily when a very low-fat food is required, but offer no advantages over a dilution of fresh whole milk. The large amount of sugar in the sweetened condensed milk makes the food so sweet that it could not be taken unless diluted to such a degree that the nourishing qualities, except for the sugar, are entirely inadequate. The 4th class in addition to the sweetening contains almost no fat, and is entirely unfitted for administration to infants.

The following table gives the percentage-composition, derived from various sources, of several of the condensed milks. These examples are drawn at random from the recorded analyses in my possession of over 50 different varieties. Some of them are now used little if at all.

In this, as in the succeeding tables, the analysis of human milk heads the list, for the convenience of comparison.

TABLE 53.—ANALYSES OF CONDENSED MILKS

	Water	Fat	Protein	Lactose	Saccharose	Mineral matter
Human Milk	87-88	3.5-4	1-1.5	6.5-7		.20
<i>Dried Milk.</i>						
Glaxo ¹	3.50	27.40	22.20	41.00	5.90
Defiance ²	4.90	27.00	26.20	36.30	5.60
Lacvitum ³	5.34	29.40	28.04	31.26	5.96
Dryco Brand ⁴	3.00	12.00	34.00	44.00	7.00
Mammala ⁴	5.00	12.00	24.00	55.00	5.00
<i>Condensed Whole Milk.</i>						
Ideal ⁵	68.27	10.10	7.36	11.03	1.85
St. Charles ⁵	66.46	9.26	10.49	12.24	1.55
Highland ⁵	68.75	9.63	9.21	10.89	1.52
First Swiss ⁶	62.15	11.38	9.90	14.44	2.10
Hollandia ⁷	57.00	9.80	11.30	18.50	3.40
<i>Sweetened Condensed Whole Milk.</i>						
Rose ⁸	23.70	11.00	9.70	14.60	38.70	2.30
Red Cross ⁵	25.97	7.93	8.91	11.93	43.77	1.49
Eagle ⁵	30.16	7.51	8.40	9.82	42.24	1.87
Anglo-Swiss ⁷	25.60	10.80	8.80	16.00	37.10	1.70
Nestlé's Condensed milk ⁸	26.30	11.50	9.70	13.00	37.50	1.90
<i>Sweetened Condensed Skimmed Milk.</i>						
Cowslip ⁹	25.68	0.71	10.35	16.85	43.09	2.48
Farm ⁹	28.48	0.60	7.90	18.76	41.77	2.04
Snake ⁹	25.88	0.96	10.64	27.38	34.07	2.56

¹ Hutchison, Food and Dietetics, 1911, 467.

² Ibid., p. 119.

³ Cautley, Sutherland's System of Diet and Dietetics, 1908, 223.

⁴ Advertisement.

⁵ Penna. Dept. of Agriculture Bulletin No. 10.

⁶ Cautley, *loc. cit.*, p. 218.

⁷ Hutchison, *loc. cit.*, p. 463.

⁸ Cautley, *loc. cit.*, p. 220.

⁹ Chapin, Infant Feeding, 1902, 79.

Condensed milks are generally diluted with from 9 to 20 parts of water before using. Assuming a dilution of 1 in 10 as convenient for calculation, we may readily see what a weak food, except for the sugar, is obtained, and can understand why condensed milk so often agrees with the digestion, but either does not support the infant, or allows it to grow flabbily fat without proper tissue, as a result of the relatively large amount of sugar present and the small amount of protein. A dilution of 1 in 10, for instance, of Eagle Brand Condensed Milk, produces a food containing only fat 0.75 per cent., protein 0.84 per cent., sugar 5.21 per cent. and salts 0.18 per cent.

II. Dextrinized (Malted) Foods.—The basis of these, often called also Liebig's Foods, is starch usually derived from wheat-flour or barley-flour which has been entirely converted into soluble carbohydrates, generally by the action of the diastase of a malt-extract. These carbohydrates consist of dextrin and maltose and intermediate substances. The foods should not give the iodine reaction for unconverted starch. To some of them milk is added in the process of manufacture. To others it is to be added when the food is mixed for the child. The quantity of sugar present is far too great to resemble in any way human milk, and it is to be noted also that it is not the natural sugar of the milk which is employed. When diluted sufficiently to reduce the sugar to a normal amount the foods are all much too weak in the percentage of fat, and often of protein as well, unless fresh milk or cream is added. These facts can be seen by examining the figures in the following table, which gives the analysis of a few of the foods of this class. There is no real need for any of these foods, inasmuch as a milk-mixture with either the addition of starch dextrinized at home or of a dextrin-maltose preparation which is not called a food, can be more conveniently, cheaply and accurately prepared in the house.

TABLE 54.—ANALYSES OF DEXTRINIZED FOODS

	Water	Fat	Protein	Soluble carbo- hydrates	Mineral matter	Remarks
Human milk.....	87-88	3.5-4	1-1.5	6.5-7	0.2	
Horlick's Malted Milk ¹ .	3.06	8.78	16.35	67.95	3.86	Milk; barley and wheat malted.
Liebe's Soluble Food ² ...	22.34	Trace	6.47	68.80	1.71	An extract of malt made from wheat.
Loefflund's Infant Food ²	25.37	Trace	4.17	68.60	1.47	Wheat and malt.
Loefflund's Peptonized Infant Milk ²	20.39	8.46	10.13	57.53	3.05	Practically a malt-extract.
Mellin's Food ¹	5.62	0.16	10.35	79.57	2.30	Wheat, malted barley ³ and bicarbonate of potash.
Laibose ¹	6.00	17.00	18.00	55.00	4.00	A combination of whole-milk with dextrinized wheat.
Justfood ¹	4.50	1.10	93.95	0.32	Dextrinized cereals.

III. Amylaceous Foods.—All of these contain unconverted starch in larger or smaller amount. In some of them malt-extract, pancreatic extract or some form of sugar has been added; but in none of them.

¹ Advertisement and information from manufacturer.

² Blauberg, Arch. f. Hyg., 1897, XXX, 125.

³ Advertisement.

TABLE 55.—ANALYSES OF AMYLACEOUS FOODS

	Water	Fat	Proteid	Total carbo- hydrates	Insoluble carbo- hydrates	Soluble carbo- hydrates	Mineral matter	Remarks
Human milk.....	87-88	3.5-4	1-1.5	6.5-7	6.5-7	0.2	
Nestlé's Infant Food ¹		5.5	14.34	74.32	15.39	58.93	2.03	A condensed milk with baked wheat-flour and cane-sugar.
Gerber's Infant Food ²	4.96	4.60	13.10	77.51	32.93	44.58	1.40	Milk sweetened, with dextrinized flour added.
Kufeke's Infant Food ³	7.60	0.88	11.17	78.51	48.01	30.50	2.07	Appears to be partly dextrinized and to have salts added.
Cannick's Soluble Food ⁴	3.12	6.26	16.32	71.06	14.44	56.62	3.02	Evaporated milk, partially malted wheat-flour and milk-sugar. ⁵
Savory and Moore's Food ⁵	5.34	1.46	10.19	81.90	54.09	27.81	0.91	Wheat-flour and malt. Much grape and cane-sugar added.
Wells, Richardson and Company's Lactated Food ⁶	7.76	1.64	11.85	75.43	36.43	39.00	2.61	Partly malted. Contains much cane-sugar and no milk.
Wagner's Infant Food ⁷	5.07	10.91	14.81	66.82	28.91	37.91	2.01	A baked flour.
Ridge's Food ⁸	8.87	1.67	13.37	74.67	66.35	8.32	0.61	A baked wheat-flour.
Imperial Granum ⁹	10.57	1.32	19.37	67.00	51.88	15.42	1.13	Wheat starch partially malted.
Thienhardt's Soluble Infant Food ¹⁰	6.87	9.58	14.37	64.30	19.77	44.53	3.56	Practically all starch.
Neave's Food ¹¹	5.03	1.7	13.2	80.4	74.27	4.71	1.09	Merely barley flour.
Robinson's Patent Barley ¹²	10.1	0.9	5.1	82.0	1.9	Merely ground oats with husks removed.
Robinson's Groats ¹³	10.4	1.6	11.3	75.0	1.7	Wheat, flour and malt. Contains unconverted starch.
Allenbury's Malted Food ¹⁴	6.5	1.0	9.2	82.3	0.5	Roasted oats made into meal. Milk-sugar added.
Milkine ¹⁵	2.74	7.12	13.37	74.82	13.63	61.19	1.32	
Hubbell's Prepared Wheat ¹⁶	5.93	1.19	14.81	77.02	60.86	16.16	0.44	
Health Food Company's Barley ¹⁷	10.92	0.89	6.98	80.23	?	0.86	
Rademann's Infant Food ¹⁸	7.67	6.00	15.10	66.43	47.89	18.54	3.69	Roasted oats made into meal. Milk-sugar added.
Benger's Food ¹⁹	11.29	1.10	10.43	76.20	66.30	9.90	0.96	Wheat flour and pancreatic extract. The large amount of starch becomes for the most part converted when the food is prepared for use.
Cereal Milk ²⁰	9.33	1.01	11.08	78.42	58.42	20.00	1.16	

¹ Ibid., p. 216.² Blauberg, Arch. f. Hyg., 1897, XXX, 125.³ Blauberg, Arch. f. Hyg., 1896, XXVII, 119.⁴ Penna, Dept. of Agriculture, Bull. No. 10.⁵ Caudley, Sutherland's System of Diet and Dietetics, 1908, 217.⁶ Leeds, New York Med. Jour., 1883, XXXVII, 449.⁷ Blauberg, Arch. f. Hyg., 1897, XXX, 125.⁸ Blauberg, Arch. f. Hyg., 1896, XXVII, 119.⁹ Penna, Dept. of Agriculture, Bull. No. 10.¹⁰ Leeds, New York Med. Jour., 1883, XXXVII, 449.¹¹ Ibid., p. 216.¹² Blauberg, Arch. f. Hyg., 1897, XXX, 125.¹³ Blauberg, Arch. f. Hyg., 1896, XXVII, 119.¹⁴ Penna, Dept. of Agriculture, Bull. No. 10.¹⁵ Caudley, Sutherland's System of Diet and Dietetics, 1908, 217.¹⁶ Ibid., p. 216.¹⁷ Blauberg, Arch. f. Hyg., 1897, XXX, 125.¹⁸ Blauberg, Arch. f. Hyg., 1896, XXVII, 119.¹⁹ Penna, Dept. of Agriculture, Bull. No. 10.²⁰ Leeds, New York Med. Jour., 1883, XXXVII, 449.

according to the analyses, is the starch entirely transformed. In the case of many of them it is admitted to be present and the food is intended to be added to milk-mixtures. In others the food is recommended without any statement regarding the presence of starch, or even with the claim that none exists in it. The accompanying table gives published analyses of a number of starch-containing foods on the market.

IV. Miscellaneous Foods.—In this class are placed a number of foods in the composition of which other substances than starch or sugar have been used, or which in some way do not properly belong to any of the previous classes. In some the proportion of cream is increased either by direct addition of this or by removal of a part of the protein, and, the food being afterward condensed, the title of condensed cream might with propriety be employed. Some are malted or peptonized; in some unconverted starch is present, and the food might be placed among the amylaceous preparations were it not for the addition of other substances. The accompanying table gives published analyses of some of them.

V. Proteid or Nitrogenous Foods.—These form a mixed group in all of which the nitrogenous element is claimed to be high as compared with other ingredients. They are not intended to be permanent substitutes for human milk and some of these fill a useful place. They may be divided into (1) beef-extracts, (2) beef-juices, (3) peptonized meat preparations, (4) other proteid foods.

1. The Commercial Beef-extracts.—These, although popularly supposed to be highly nourishing from the amount of protein contained, possess in reality but a small percentage of this, while the extractive matter and salts are present in large amount. They are prepared under the influence of water, heat and pressure. Some of the muscle-fibre which remains is added and the liquid then evaporated. Only those in which the fibre has been thus used contain an amount of protein worth considering. A table of analyses of a number of them published by Hutchison¹ shows a total percentage of soluble protein varying in round numbers from 3 to 33 per cent. As only small doses are customarily given, or could, indeed, be tolerated on account of the excess of mineral matter and extractives, the amount of protein received by the infant is in reality trifling; given in doses of a few drops, as is often done, either alone or in addition to the milk in the bottle.

2. Beef-juices.—These consist of expressed juice or serum of the beef. According to analyses of a number of the commercial preparations, as published by Hutchison,² the percentage of coagulable protein varies from 17 per cent. to 0.3 per cent., nearly all of those in his list containing not over 5 per cent. Although some of them are richer in protein than is freshly prepared beef-juice, yet, as in the case of the beef-extracts, the salts and extractive matter are generally present in too large an amount to permit of the giving them in sufficient quantity to be of real benefit. My preference has always been decidedly for the freshly prepared beef-juice (p. 150).

3. Beef-powders and Peptonized Beef-preparations.—Some of these are extensively employed for temporary use in infant-feeding and infant-therapeutics. Prominent among them are a number whose nitrogenous value is slight and whose percentage in alcohol is high. On account of their comparatively pleasant taste they may be used in place of other

¹ Food and Dietetics, 1911, 96.

² *Loc. cit.*, 100.

TABLE 56.—ANALYSES OF MISCELLANEOUS FOODS

	Water	Fat	Protein	Total carbo- hydrates	Insoluble carbo- hydrates	Soluble carbo- hydrates	Mineral matter	Remarks
Human Milk	87-88	3.5-4	1-1.5	6.5-7	0.2	
Voltmer's Mother's Milk ¹	22.10	9.08	13.26	51.52	51.52	4.04	Milk boiled. Water, sugar, cream and salts added to form average percentages of human milk. Peptonized. Evaporated.
Biedert's Ramogen ²	16.5	7.0	34.65	34.65	1.5	Milk evaporated. Cream, cane-sugar and an alkali added. Sterilized.
Muller's Infant Food ¹	4.28	6.47	14.48	72.54	47.62	24.92	2.28	Wheat-flour, milk, eggs, milk-sugar, wheat-gluten.
Allenbury's Food No. 1 ³	5.7	14.0	9.7	66.85	66.85	3.75	Desiccated milk; excess casein removed; vegetable-albumin, milk-sugar and cream added.
Allenbury's Food No. 2 ³	3.9	12.3	9.2	72.1	72.1	3.50	Like No. 1, but with addition of some malted flour.
Liebig's Maltol-leguminose mit Zucker ⁴	8.44	1.37	20.34	65.10	48.19	16.91	2.97	Leguminose flour partly malted.
Frane Food Diet ⁵	5.0	1.2	13.4	79.4	1.0	Baked flour; cane-sugar; extract of bran. Itch in protein and unaltered starch.
Lacto preparata ⁵	5.80	12.35	14.51	63.68	63.68	3.66	Addition of milk-sugar instead of malted starch or cane-sugar.
Freiich's Soluble Infant Food ¹	8.44	5.97	12.98	69.82	27.59	42.23	1.67	Finely dextrinized leguminous flour.
Gartner's Mother-milk ⁶	3.05	2.09	6.0	6.0	Milk centrifuged. To the cream-portion milk-sugar and an alkali are added.
Eskay's Albumenized Food ²	1.70	3.52	6.70	87.02	31.20	55.82	0.99	Digested cereals combined with egg-albumen. Milk and malted flour. Apparent casein removed, or at least with it.
Loefflund's Condensed Cream ⁷	20.2	23.0	5.0	50.0	50.0	1.8	Infant food, with milk and cream. Cream and milk-sugar added. Three formulæ of different strengths.
Backhaus' Infant Milk ⁸	I. 88.93 II. 80.91 III. 87.93	3.16 3.22 3.40	1.32 1.74 2.92	6.04 5.71 4.99	6.04 5.71 4.99	0.55 0.42 0.71	Probably chiefly vegetable albumin. Almonds and other nuts and sugar used in its preparation.
Lahmann's Vegetable Milk ⁹	24.44	24.60	7.05	43.10	1.15	Wheat-flour partly malted; yellow of egg; cocoa-butter.
Odda ¹⁰	5.8	6.49	14.57	71.84	2.10	Buttermilk, wheat-flour, cane-sugar prepared as for the ordinary buttermilk mixture.
Biedert's Buttermilk Conserve ²	0.6	9.6	34.5	4.5	30.0	2.58	Milk condensed to $\frac{1}{4}$ its volume.
Hoos' Maltose Buttermilk ¹¹	10.5	33.5	4.5	29.0	1.7	Very similar to the last, but a dextrin-maltose employed.

¹ Blauberg, Arch. f. Hyg., 1896, XXVII, 119.² Advertisement.³ Hutchison, Food and Dietetics, 1911, 468.⁴ Blauberg, Arch. f. Hyg., 1897, XXX, 125.⁵ Advertisement (Ref. M. and G. C. Lincoln, Artificial Feeding in Children, 1916).⁶ Poole (Fischer, New York Med. Rec., 1897, Dec. 11).⁷ Biedert, Kinderernähr. im Sauglingsalter, 1900, 196.⁸ Hartung, Jahrb. f. Kinderh., 1902, LV, 676.⁹ Hock, Wien. med. Wochenschr., 1896, 435.¹⁰ Mering, Therap. Monatsh., 1902, 175.¹¹ Statement by manufacturer.

alcoholic stimulants in infancy and childhood. The following analyses are given by Harrington.¹

TABLE 57.—ALCOHOLIC STRENGTHS OF LIQUID BEEF-PREPARATIONS

	Alcohol by volume, per cent.	Total solids, per cent.
Liquid Peptonoids.....	23.03	14.91
Panopeptone.....	18.95	17.90
Hemapeptone.....	10.60	19.54
Nutritive Liquid Peptone.....	14.81	15.20
Hemaboloids.....	15.81	6.36
Tonic Beef.....	15.58	18.16
Mulford's Predigested Beef.....	19.72	10.39

4. Other Proteid Foods.—Among other preparations the following may be mentioned:

*Mosquera's beef-jelly*² which contains 28.63 per cent. of protein in a partially digested state.

Somatose.—A meat-powder completely peptonized and containing over 80 per cent. of protein (Neumann).³ Another form, Milk Somatose, is derived from milk and contains about 70 per cent. of protein (Neumann). Somatose is often useful as an addition to milk.

Dry Peptonoids.⁴—This contains 40 per cent. of protein, the remainder of its nutritive value depending entirely on carbohydrates (51.5 per cent.).

Tropon.—A powder rich in protein (90.44 per cent. (Fröhner and Hoppe)⁵ derived both from animal and vegetable sources. It is not predigested.

Roborat.—A food in powdered form made from various grains and containing about 95 per cent. of vegetable-albumin (Sommerfeld).⁶

Hygama.—A powder giving 21.93 per cent. of nitrogenous substance as well as a high percentage of carbohydrate, most of which is in soluble form. It is flavored with cocoa (Kraus).⁷

Plasmon represents in powdered form 75 to 80 per cent. of albumin derived from milk, with the addition of 5 to 7 per cent. of carbonate and bicarbonate of soda. It is in reality an alkali-casein (Laves).⁸

Nutrose.—A powdered food formed of a soda-combination with casein. It possesses 73.68 per cent. of albumin and 11.67 per cent. of water (Neumann).⁹

Soson.—A meat-derivative containing 92.5 per cent. albumin (Neumann).¹⁰

Eucasin.—A powdered preparation of casein consisting of 77.60 per cent. of nitrogenous material (König).¹¹

Forsan.—A powdered product made of beef-blood. It contains 84 per cent. of nitrogenous material (König).¹²

¹ Boston Med. and Surg. Journ., 1903, Mar. 12, 283.

² Wylie, Foods and Their Adulterations, 1911, 566.

³ Münch. med. Wochenschr., 1893, 395.

⁴ Advertisement, Arlington Chemical Co.

⁵ Münch. med. Wochenschr., 1899, 52.

⁶ Archiv für Kinderheilk., 1903, XXXVI, 341.

⁷ Therap. Monatsh., 1902, XVI, 635.

⁸ Münch. med. Wochenschr., 1900, XLVII, 1339.

⁹ Loc. cit., 72.

¹⁰ Loc. cit., 106.

¹¹ Nahrungs- u. Genussmittel, 1904, II, 539.

¹² Loc. cit.

*Albulactin*¹ is a grey-white powder consisting chiefly of lactalbumin, and having 83.60 per cent. of protein. It may be added in small amounts to mixtures of cow's milk.

Larosin.—This preparation, devised by Stöltzner,² is a pure calcium caseinate containing the equivalent of 2.5 per cent. of calcium oxide. It was devised to simplify the process of preparing casein milk. When 2 per cent. of it is added to equal parts of milk and water, it gives a mixture containing fat 1.75 per cent., sugar 2.25 per cent., protein 3.45 per cent., phosphoric oxide 0.122, calcium oxide 0.136. The mixture has the advantage that it can be boiled. It is frequently an excellent and convenient substitute for casein milk.

Hoos' Albumin Milk.³—This consists of a dried casein-milk of a strength and composition to reproduce when diluted, the original formula of Finkelstein. As supplied in the can it contains protein 38 per cent. fat 30 per cent, milk-sugar 19 per cent., salts 5 per cent. As with the freshly prepared casein-milk, sugar in some form will need to be added after the infant's disturbances of digestion are relieved.

Sanatogen (Treat).⁴—This is a preparation containing about 90 per cent. of casein, with sodium-glycerophosphate and a small amount of unidentified nitrogenous compound. It would appear to possess no advantage over the very much cheaper commercial casein.

¹ Advertisement, Analytical Report, Lancet, 1911, I, 34.

² Münch. med. Wochenschr., 1913, LX, 291.

³ Advertisement and information received from manufacturer.

⁴ Journ. Amer. Med. Assoc., 1914, LXIII, 1831.

CHAPTER VII

DIET AFTER THE FIRST YEAR

DIET FROM 12 TO 18 MONTHS

By the time the infant is 1 year old it will have been weaned and be fed upon cow's milk nearly or quite undiluted, and it should have commenced to take a certain amount of starchy material in the milk (p. 152). From this age its diet is gradually extended. It is better first to increase the amount of starch by having the child learn to eat stale bread or toast moistened with milk and fed from a spoon; a well-cooked porridge of farina, oatmeal, hominy or wheaten grits, arrowroot, sago, tapioca, etc., one not of oatmeal being the first to be tried; or some form of the numerous excellent farinaceous breakfast foods on the market, those which require cooking being much to be preferred. All the porridges must be seasoned with salt in their preparation or before use, a small amount of milk poured over them, and a little cane-sugar added, if necessary, to make the infant eat them. They may be made from the grain or from the flour, using about 4 level tablespoonfuls to the pint of milk or water. The boiling must be slow and continue for from 3 to 6 hours for broken grain, and at least $\frac{1}{2}$ hour for flours. The porridge may with advantage be employed for the midday meal, the other meals still consisting of milk. Very shortly it may instead be given for breakfast, and for dinner the child may receive bread or well-boiled rice, moistened with beef-juice or with dish gravy as free as possible from fat. Soft-boiled eggs may be tried at about the age of 15 months, taking pains that the white is not firmly coagulated, and remembering that many children do not tolerate eggs at all well until after the 18th or 20th month, and others not even then unless given them only occasionally. All through this period milk should remain the principle article of diet, and care with regard to its bacterial content continued. Yet precautions must be taken that milk is not practically the only food ingested, but that as the amount of solid food is increased that of the milk be reduced somewhat. The more milk there is poured upon the porridge, the less should be drunk from the cup. A total of from 24 to 32 oz. should usually be the outside daily limit. The milk may be slightly or not at all diluted, or may have a small amount of some cereal jelly added to each bottle. If it is from Jersey cattle the removal of some of the cream is advisable. Very many infants in the 2d year do not tolerate undiluted milk, especially if it is rich in cream. Somewhere between 12 and 18 months of age the infant should have been taught to take most of the milk from a cup or glass, inasmuch as it is very difficult to combine the eating of solid food at meal-time with the drinking of milk from a bottle. The milk from the bottle will be taken without pause and the desire for solid food interfered with; or if the bottle is given last, the child takes too much nourishment for one meal. An exception may be made, if desired, as regards the bottle before the morning nap and that at 9 or 10 p.m. In the case of many infants the juice from a small orange, or that of well-stewed

prunes, may properly be added to the dietary. These aid in avoiding constipation, and prevent the development of scurvy, but neither of them can be considered indispensable articles of diet for healthy children. Orange-juice may be given an hour before one of the feedings. Sometimes it is useful to begin it toward the end of the 1st year or even before this.

The following list may be followed as a guide for the feeding of children from 12 to 18 months of age. A numbered selection of dietaries is given.

TABLE 58.—DIET FROM 1 YEAR TO 18 MONTHS

Breakfast (6 to 7 A.M.): (1) 8 to 10 oz. of milk with stale bread, toast or zweiback broken into it. (2) 2 to 3 tablespoonfuls of well-cooked oatmeal, arrowroot, wheaten grits, hominy grits, farina, etc., or one of the numerous good breakfast foods on the market, not of the ready-to-serve class, made into a well-cooked unstrained porridge, and with 6 to 8 oz. of milk poured over it. (3) A soft-boiled or poached egg (after 15 months) with stale bread thinly buttered, and a cup of milk.

Second Meal (10 A.M.): 8 or 10 oz. of milk from a cup or bottle.

Dinner (1:30 to 2 P.M.): (1) Stale bread moistened with dish-gravy (no fat), beef-tea, or 1 to 2 oz. of beef-juice; a cup of milk. (2) Rice or grits moistened in the same way; 6 to 8 oz. of milk. (3) A soft-boiled egg and stale bread thinly buttered; 6 to 8 oz. of milk. Sago, tapioca, or rice pudding (no raisins), junket, or cornstarch in small quantities as dessert with any of these diets.

Fourth Meal (5 to 6 P.M.): 8 to 10 oz. of milk; or some bread and milk.

Fifth Meal (9 to 10 P.M.): 8 to 10 oz. of milk from a cup or bottle.

This list is intended as suggestive only. It is also one to which the child must grow accustomed very gradually, and it represents the *extreme* of what may be given rather than what must necessarily be eaten. Many infants do well with a dietary very much weaker than this. Inasmuch as the daily amount of milk is now restricted, care must be taken that the child receive water. The milk should not be used as a drink to replace this in order to quench thirst. If it is desired, a cup of broth may take the place of milk at dinner; but there is usually, in my experience, no advantage in this, and it can well be dispensed with until after the age of 18 months. The number of teeth which have been cut should have little influence upon the selection of the food during this period, since the infant does but little masticating in any event. The weight and general condition are better guides.

DIET FROM 18 MONTHS TO 2 YEARS]

During this period but little change is made in the dietary. Milk is still a standby, and should have the same precautions against contamination taken with it as earlier in the infant's life, but the quantity is being diminished by the employment of more solid food. Potatoes may be given occasionally for dinner, although the starch of this vegetable seems often less digestible than some other varieties. Finely minced meat is also advisable. Orange-juice, well-stewed prunes mashed through a sieve, and well-baked apple taken with little or no sugar, are useful additions. It is the growing custom to begin the employment of green vegetables by this time; sometimes even earlier. This can be done cautiously and is often beneficial, especially where a tendency to constipation exists. The vegetables should be thoroughly cooked, or, better, well-steamed to favor the retention of the salts (Bartlett),¹ and then rubbed through a fine sieve. Some children assimilate food of this sort

¹ Arch. of Pediat., 1917, XXIV, 436.

readily; others find difficulty in digesting it even at a decidedly later period. The fifth meal is no longer desirable. The following list is, like the last, a guide only.

TABLE 59.—DIET FROM 18 MONTHS TO 2 YEARS

Breakfast (7 A.M.): (1) 8 oz. of milk with a slice of buttered bread or toast, or a soda, oatmeal or other unsweetened biscuit. (2) A soft-boiled or poached egg, with bread and butter and 6 to 8 oz. of milk. (3) Porridge as described in the previous list.

Second Meal (10 A.M.): (1) Bread broken in milk. (2) Bread and butter or a soda or other biscuit with 6 to 8 oz. of milk.

Dinner (2 P.M.): (1) Boiled rice or a baked potato mashed and moistened with dish-gravy or beef-juice; or from $\frac{1}{2}$ to 1 tablespoonful of spinach, string-beans, peas, asparagus tips or carrots; 6 oz. of milk. (2) Six ounces of mutton or chicken broth with barley or rice in it and the meat-fibre remaining (see p. 151); some bread and butter, and some sago or rice-pudding. (3) A small portion (2 or 3 teaspoonfuls) of minced white meat of chicken, turkey, rare beef or beef-steak, lamb, mutton, or fish; bread and butter; 8 oz. of milk. A baked apple, or a few stewed prunes pressed through a colander may be allowed as dessert with some of these dietaries at the meals when green vegetables are not given.

Fourth Meal (6 P.M.): (1) Bread and milk. (2) Milk with soda or other biscuit, toast, zweiback, or bread and butter. (3) Two or 3 tablespoonfuls of a cereal-porridge with 8 oz. of milk.

DIET FROM 2 TO 3 YEARS

Meat which is merely cut up, not scraped or minced, is now added to the list. Stewed fruit in greater quantities can be used, and many fresh fruits if ripe are excellent, such as peaches, grapes without seeds and raspberries. Bananas should be given not at all, and strawberries very cautiously and only if sweet and quite ripe. There is generally not the same need for specially germ-free milk of definite percentage-strength as existed earlier in life, although precautions should still be taken to obtain a good and pure article. A total of 24 ounces daily should be sufficient and it should not be rich in fat. It should have the chill removed, but need not be actually warm. Selected green vegetables should now certainly be added to the dietary, if they have not been given previously.

The following list is a guide for this period.

TABLE 60.—DIET FROM 2 TO 3 YEARS

Breakfast (7 to 8 A.M.): (1) A small portion of beef-steak, with 2 to 3 tablespoonfuls of farina, oatmeal, hominy-grits, wheaten grits, corn-meat, or other cereal-porridge with 6 or 8 oz. of milk upon it. (2) A soft boiled egg; bread and butter, and 6 or 8 oz. of milk.

Second Meal (11 A.M.): 8 oz. of milk, with bread and butter or with a soda or other biscuit. (2) Bread and milk. (3) Six ounces of chicken or mutton broth.

Dinner (2 P.M.): Roasted fowl, mutton, or beef cut fine; mashed baked potato, rice, or macaroni, with butter or dish-gravy on it; bread and butter, a green vegetable of the sort and prepared in the manner described. As dessert, tapioca, sago, or rice-pudding, junket, or some of the fruits mentioned.

Supper (6 P.M.): (1) Bread and butter. (2) Eight ounces of milk, with soda or similar biscuit, or with bread and butter. (3) Two to 4 tablespoonfuls of a cereal-porridge, with 8 oz. of milk. (4) A soft-boiled egg with bread and butter. It makes a pleasant variation to give the egg for supper on some days and for breakfast on others; but it should not be allowed twice a day.

Again must be emphasized the statement made, that these tables are a guide only, not an absolute rule, and that they represent rather the extreme than the requirements of the dietary.

No increase of diet should be made in the midst of a heated term unless it is absolutely necessary. Indeed, it is better in very hot weather to

return to one suitable for a much younger child, and to let it be largely of milk, diluted more than usual. It is sometimes advisable to use a small amount of cream upon the cereal-porridge. In my own experience healthy children are better without it. Very many children exhibit great difficulty in digesting much starchy food; and others, as stated, show some degree of intolerance for egg. In such cases meat may occasionally be allowed for breakfast as well as for dinner. As a rule, however, meat should be given but once a day. The ready-to-serve cereals may be given occasionally, but should be avoided as a steady diet. At the outbreak of any acute illness, especially of the digestive apparatus, the diet should be very greatly reduced in variety and amount. Of course, the existence of a chronically weak digestion modifies greatly all the schemes for diet which have been detailed. There is no good foundation for the popular fear of the "second summer," provided the diet be watched then with the same care as in the first summer. A child which has been breast-fed has had the advantage in its first summer of being upon its natural food; and it is on this account that the "second summer" has acquired a bad reputation.

Thorough mastication and slowness of eating must be taught as early as possible, but it is difficult to obtain this. Neither before the age of 3 years nor after it should food be allowed between meals, unless there is actual hunger, and then the habit of irregular eating must not be allowed to form. Allowing the child to go hungry on a few occasions will often get it in the way of eating more heartily at its regular meal-times. During temporary loss of appetite eating should not be urged, since the child may be merely following Nature's demands for a rest of the digestive organs. Care should be taken, too, not to put too much sugar upon cereal-gruels. A very little may be used, but it is better to accustom the child to take them without sugar.

The meals, up to about the age of $2\frac{1}{2}$ years, are best given in the nursery, but from this time the child can well sit at the table in its high chair, or at a small table close by, provided that it never be given articles of food not suited to its age.

DIET FROM 3 TO 6 YEARS

From 3 years onward the dietary is decidedly increased in variety, approaching more closely, although gradually, that of the adult. All rich, highly seasoned dishes are to be avoided. Milk, not rich, must still form a very prominent article of diet, and should continue to do so up to the age of 5 or 6 years at least. Cream should be employed with great caution and in small quantity. The heartiest meal should be in the middle of the day, and the supper should be light. Meat may be given once or twice a day, depending upon the needs of the child and the character of its digestion.

The following lists may be found useful as a guide.

TABLE 61.—FOODS PERMITTED

Meats.—Broiled beef-steak, lamb chops and chicken; broiled liver; roasted or boiled beef, mutton, lamb, chicken and turkey; broiled or boiled fish.

Eggs.—Soft-boiled, poached, scrambled, omelette.

Cereal Foods.—Light and not too fresh wheaten and Graham bread, toast, zwieback; plain unsweetened biscuit, as oatmeal, soda, water, etc.; hominy grits, wheaten grits, corn-meal, barley, rice, oatmeal, macaroni, etc.

Soups.—Plain soup and broth of nearly any kind.

Vegetables.—White potatoes, boiled onions, spinach, peas, carrots, asparagus

except the hard parts, string and other beans, salsify, lettuce, stewed celery, young beets, arrowroot, tapioca, sago, etc.

Fruits.—Nearly all if stewed and sweetened; of raw fruits, used moderately, peaches are one of the best; pears; well-ripened and fresh raspberries; blackberries; grapes without the skin and seeds; oranges without the rind.

Desserts.—Light puddings, as rice pudding without raisins, bread pudding, etc., plain custards, wine-jelly, junket, and occasionally ice-cream.

FOOD TO BE TAKEN WITH CONSIDERABLE CAUTION

Kidney, oysters, muffins, hot rolls, sweet potatoes, baked beans, squash, turnips, parsnips, egg-plant, stewed tomatoes, green corn, cherries, plums, raw apples, strawberries, blueberries, gooseberries, currants.

FOODS TO BE AVOIDED

Fried food of any kind, griddle-cakes, pork, sausage, ham, goose, veal, corned-beef, pastry, salt fish, highly seasoned foods, all heavy, doughy, or very sweet puddings, unripe, sour or wilted fruit, bananas, pineapples, cucumbers, radishes, raw celery, raw tomatoes, cabbage, cauliflower, nuts, candies, sweet cakes, preserved fruits, jams, tea, coffee, alcoholic beverages.

After the age of 5 or 6 years the diet may be still further increased in variety, being almost similar to that suitable for adults; but care must be followed, and close supervision given up to the age of puberty.

AMOUNT OF FOOD-ELEMENTS AND NUMBER OF CALORIES REQUIRED AFTER THE FIRST YEAR

A knowledge of the percentage-composition and caloric value of the foods given after the 1st year is less often required than in infancy, but still often very serviceable. This is particularly true when indigestion of some form develops, or when the question of over-feeding or under-feeding arises. The caloric needs of children after the age of 1 year have not been very extensively studied. The matter is reviewed by Locke,¹ as also by Knox,² based upon studies by Camerer,³ Sommerfeld⁴ and others. The total amounts of the different ingredients necessary vary decidedly, this depending in part upon the fact that the food-elements are to a considerable extent interchangeable, but that if, for instance, a larger amount of fat is given the total quantity of food required is less, on account of the greater caloric value of this. The average amounts given by Sommerfeld, based upon various collected analyses are shown in the following table:

TABLE 62.—DAILY QUANTITY OF THE DIFFERENT FOOD-ELEMENTS REQUIRED

Age	Protein, grams	Fat, grams	Carbohydrates, grams
2-4 years	40-64	32-62	110-205
5-7 years	50-58	30-43	145-197
8-10 years	60-88	30-70	220-250
10-11 years	68-86	44-85	211-270

In round numbers he places the needs as:

From 2 to 4 years, protein 50 grams (1.76 oz. Av.), fat 50 grams (1.76 oz. Av.), carbohydrates 140 grams (4.94 oz. Av.).

From 5 to 8 years, protein 80 grams (2.82 oz. Av.), fat 65 grams (2.29 oz. Av.), carbohydrates 220 grams (7.76 oz. Av.).

From 8 to 12 years, protein 85 grams (3 oz. Av.), fat 80 grams (2.82 oz. Av.), carbohydrates 275 grams (9.70 oz. Av.).

¹ Boston Med. and Surg. Journ., 1912, CLXIX, 702.

² Journ. Amer. Med. Assoc., 1916, LXVII, 432.

³ Der Stoffwechsel des Kindes, 1896.

⁴ Pfandler und Schlossmann, Handb. der Kinderh., 1903, I, 401.

The actual amount of food needed increases steadily as the child grows older, but the relative amount as compared with the body-weight; *i.e.* the energy-quotient, decreases steadily. This is dependent in part upon the diminished ratio between the surface and the body-weight, and in part upon the relatively more rapid growth in the younger subjects. Taking the weight as our guide, the caloric requirements for the 1st year have already been detailed (p. 53). For the age of 2 years and upward, as determined by Camerer,¹ they may be seen in the following table given by Sommerfeld.²

TABLE 63.—DAILY TOTAL AMOUNT OF FOOD-ELEMENTS IN GRAMS, AND OF THE CALORIES, PER KILOGRAM OF BODY-WEIGHT

Age in years	Sex	Total food, grams	Water, grams	Protein, grams	Fat, grams	Carbohydrates, grams	Calories per kilogram of body-weight
2-4	Each.....	93.1	75.3	3.6	3.1	9.2	75.3
5-7	Girls.....	84.4	67.4	3.0	1.9	10.7	69.0
	Boys.....	84.3	66.6	3.5	2.5	10.9	76.6
7-10	Girls.....	75.5	59.0	2.7	1.3	9.9	59.2
	Boys.....	70.8	55.5	2.8	1.3	10.4	61.0
10-14	Girls.....	54.0	41.4	2.1	1.4	8.4	51.4
	Boys.....	56.1	44.4	2.5	1.0	7.7	47.3

All but the protein may be varied, but this should be approximately the amount given in the table. About 30 per cent. of the food should be of animal origin, and about 50 per cent. of the protein should be of this nature, the rest being of vegetable derivation (Camerer). An examination of the table shows that the protein should be from 20 to 25 per cent. of the total diet excluding water.

PERCENTAGE-COMPOSITION AND CALORIC VALUE OF VARIOUS FOODS IN INFANCY AND CHILDHOOD

In Table 64 may be found the percentage-composition and caloric value of a number of dietary articles likely to be employed in infancy and childhood. They are taken, with a few exceptions indicated by an asterisk, from the "Composition of American Food-materials" by Atwater and Bryant.³ The edible portion of the food is that referred to in nearly all instances, and always unless otherwise mentioned, when the letters "A. P." (as purchased) are appended, as by the authors. All foods are raw unless otherwise stated. The equivalents of the caloric values for kilograms are given in addition to the avoirdupois estimations of Atwater and Bryant. The caloric values are given in round numbers. The percentage-figures for human milk are those very commonly adopted, and those for top milk are the approximate averages derived from a number of sources, and the caloric equivalents for these are calculated by Fraley's formula (p. 123). In the measuring of milk and other liquids, as expressed in ounces, since 1 pint of water weighs practically 1 pound avoirdupois, the difference between the fluid ounce and the ounce avoirdupois,

¹ *Loc. cit.*

² *Loc. cit.*, 402.

³ U. S. Dept. Agriculture, Office of Experimental Station, Bull. 28, Revised Edit., 1906.

dupois is slight enough to be disregarded, both as to percentage-composition and caloric value. A smaller list of the caloric values of certain foods as prepared for use will be given later. (See p. 177.)

Caloric Value of Cooked Foods.—The caloric estimation of cooked articles of diet can be only approximate, depending upon the manner in which the cooking is done. This applies with especial force to the porridges, since the proportions of water and of cereal substance vary greatly according to the recipe employed. Table 65 (p. 177) is abstracted from the long and useful list given by Locke.¹ For further details regarding the method of preparation the reader is referred to his book upon the subject.

TABLE 64.—PERCENTAGE-COMPOSITION AND CALORIC EQUIVALENTS OF VARIOUS FOODS

Food material	Water, per cent.	Protein, per cent.	Fat, per cent.	Carbo- hydrate, per cent.	Ash, per cent.	Calories per avoirdupois		Calories per kilogram or litre
						oz.	lb.	
<i>Milk Foods.</i>								
*Human milk.....	87.0	1.5	4.0	7.0	0.2	21	330	728
Cow's milk (average).....	87.0	3.3	4.0	5.0	0.7	20	326	719
Skimmed milk; commercial fat-free.....	90.5	3.4	0.3	5.1	0.7	11	170	375
*Top milk, 32 per cent. fat..	2.5	32.0	3.4	0.7	87	1398	3082
*Top milk, 20 per cent. fat..	2.9	20.0	3.9	0.7	59	936	2064
*Top milk, 16 per cent. fat..	3.05	16.0	4.2	0.7	48	769	1695
*Top milk, 12 per cent. fat..	3.2	12.0	4.3	0.7	39	630	1389
*Top milk, 10 per cent. fat..	3.3	10.0	4.4	0.7	35	554	1221
*Top milk, 7 per cent. fat...	3.4	7.0	4.45	0.7	27	437	963
Buttermilk.....	91.0	3.0	0.5	4.8	0.7	10	165	364
Whey.....	93.0	1.0	0.3	5.0	0.7	8	125	276
Butter.....	11.0	1.0	85.0	...	3.0	225	3605	7949
<i>Meats.</i>								
Beef, lean ribs.....	67.9	19.6	12.0	...	1.0	54	870	1918
Beef, lean round.....	70.0	21.3	7.9	...	1.1	46	730	1609
Beef, roasted, A.P.....	48.2	22.3	28.6	...	1.3	101	1620	3572
Beef-steak: round, cooked, fat removed, A.P.....	63.0	27.6	7.7	...	1.8	53	840	1852
Beef-steak, loin broiled....	54.8	23.5	20.4	...	1.2	81	1300	2866
Beef-juice.....	93.0	4.9	0.6	...	1.5	7	115	254
Beef-liver.....	71.2	20.4	4.5	1.7	1.6	38	605	1334
Mutton, leg, lean.....	67.4	19.8	12.4	...	1.1	56	890	1962
Lamb-chop, broiled.....	47.6	21.7	29.9	...	1.3	104	1665	3671
Fish, Halibut.....	75.4	18.6	5.2	...	1.0	35	565	1246
Fish, Mackerel.....	73.4	18.7	7.1	...	1.2	40	645	1422
Bacon, smoked, lean.....	31.8	15.5	42.6	...	11.0	130	2085	4597
Fowl.....	63.7	19.3	16.3	...	1.0	65	1045	2304
Capon, cooked.....	59.9	27.0	11.5	...	1.3	61	985	2172
Turkey.....	55.5	21.1	22.9	...	1.0	85	1360	2998
Turkey, roast.....	52.0	27.8	18.4	...	1.2	81	1295	2855
<i>Eggs.</i>								
Eggs, white boiled.....	73.7	13.4	10.5	...	1.0	45	720	1587
Eggs, white boiled.....	86.2	12.3	0.2	...	0.6	16	250	551
Eggs, yolk boiled.....	49.5	15.7	33.3	...	1.1	107	1705	3759
<i>Sugar and Starch.</i>								
Cane-sugar, granulated	100.0	...	116	1860	4101
*Milk-sugar.....	116	1860	4101
Arrowroot.....	2.3	97.5	0.2	113	1815	4002
Cornstarch.....	90.0	...	105	1675	3693

¹Food Values, 1916.

TABLE 64.—PERCENTAGE-COMPOSITION AND CALORIC EQUIVALENTS OF VARIOUS FOODS (*Continued*)

Food material	Water, per cent.	Protein, per cent.	Fat, per cent.	Carbo- hydrate, per cent.	Ash, per cent.	Calories per avoirdupois		Calories per kilo- gram
						Oz.	Lb.	
<i>Sugar and Starch (Cont'd.).</i>								
Sago.....	12.2	9.0	0.4	78.1	0.3	102	1635	3605
Tapioca.....	11.4	0.4	0.1	88.0	0.1	103	1650	3638
<i>Cereals.</i>								
Barley, pearled.....	11.5	8.5	1.1	77.8	0.1	103	1650	3638
Barley-flour.....	11.9	10.5	2.2	72.8	2.6	103	1640	3616
Oat-meal.....	7.3	16.1	7.2	67.5	1.9	116	1860	4101
Oats, various preparations.	7.9	16.3	7.3	66.8	1.7	116	1855	4090
Rice.....	12.3	8.0	0.3	79.0	0.4	102	1630	3594
Wheat, cracked.....	10.1	11.1	1.7	75.5	1.6	105	1685	3715
Wheat-flour (average)....	12.0	11.4	1.0	75.1	0.5	103	1650	3638
Wheat, whole, flour.....	11.4	13.8	1.9	71.9	0.9	105	1675	3693
Wheat, farina.....	10.9	11.0	1.4	76.3	0.4	105	1685	3715
Wheat, macaroni.....	10.3	13.4	0.9	74.1	1.3	104	1665	3671
Wheat-bread, average....	35.3	9.2	1.3	53.1	1.1	76	1215	2679
Whole wheat-bread.....	38.4	9.7	0.9	49.7	1.3	71	1140	2513
Bread, Graham.....	35.7	8.9	1.8	52.1	1.5	76	1210	2668
Wheat-bread, toast.....	24.0	11.5	1.6	61.2	1.7	89	1420	3131
Zwieback.....	5.8	9.8	9.9	73.5	1.0	123	1970	4343
Rolls (average).....	29.2	8.9	4.1	56.7	1.1	87	1395	3075
Corn-meal.....	11.6	8.4	4.7	74.0	1.3	108	1730	3814
Corn-bread.....	38.9	7.9	4.7	46.3	2.2	75	1205	2657
Corn-hominy.....	11.8	8.3	0.6	79.0	0.3	103	1650	3638
Crackers, various average.	6.8	10.7	8.8	71.9	1.8	119	1905	4200
<i>Vegetables.</i>								
Asparagus, A.P.....	94.0	1.8	0.2	3.3	0.7	7	105	231
Beans, Lima.....	68.5	7.1	0.7	22.0	1.7	36	570	1257
Beans, string.....	89.2	2.3	0.3	7.4	0.8	12	195	430
Beets.....	87.5	1.6	0.1	9.7	1.1	13	215	474
Carrots.....	88.2	1.1	0.4	9.3	1.0	13	210	463
Celery.....	94.5	1.11	0.1	3.3	1.0	5	85	187
Lettuce.....	94.7	1.2	0.3	2.9	0.9	6	90	198
Onions.....	87.6	1.6	0.3	9.9	0.6	14	225	496
Peas.....	74.6	7.0	0.5	16.9	1.0	29	465	1025
Potatoes.....	78.3	2.2	0.1	18.4	1.0	24	385	848
Potatoes, sweet.....	69.0	1.8	0.7	27.4	1.1	36	570	1257
Spinach.....	92.3	2.1	0.3	3.2	2.1	7	110	243
Squash.....	88.3	1.4	0.5	9.0	0.8	13	215	474
<i>Fruits.</i>								
Apples.....	84.6	0.4	0.5	14.2	0.3	18	290	639
Blackberries.....	86.3	1.3	1.0	10.9	0.5	17	270	595
Dates, pressed.....	15.4	2.1	2.8	78.4	1.3	101	1615	3561
Figs, pressed.....	18.8	4.3	0.3	74.2	2.4	92	1475	3252
Grapes.....	77.4	1.3	1.6	19.2	0.5	28	450	992
Oranges.....	86.9	0.8	0.2	11.6	0.5	15	240	529
Peaches.....	89.4	0.7	0.1	9.4	0.4	12	190	419
Pears.....	84.4	0.6	0.5	14.1	0.4	18	295	650
Prunes, dried.....	22.3	2.1	...	73.3	2.3	88	1400	3087
Raspberries, red.....	85.8	1.0	...	12.6	0.6	16	255	562

TABLE 65.—CALORIC VALUE OF DEFINITE PORTIONS OF FOODS PREPARED FOR USE

Foodstuffs	Quantity	Weight, grams	Total calories
<i>Meats.</i>			
Beef-juice.....	4 oz.	120	31
Beef, roast.....	1 slice	100	357
Beef, roast (lean).....	1 slice	100	111
Steak, tenderloin.....	1 slice	100	286
Chicken, roast.....	1 slice	100	181
Lamb chop (with bone).....		100	367
Mutton, roast.....	1 slice	75	234
Mackerel, boiled.....		70	104
Halibut, boiled.....		100	121
<i>Soups.</i>			
Cream (various sorts).....	4 fl. oz.	125	70-162
Consommé.....	4 fl. oz.	120	14
Bouillon.....	4 fl. oz.	120	13
Beef-soup.....	4 fl. oz.	120	32
Bean-soup.....	4 fl. oz.	120	78
Chicken-soup.....	4 fl. oz.	120	72
<i>Vegetables.</i>			
Beans, string.....	2 hp. tbsp.	60	13
Beans, lima.....	2 hp. tbsp.	80	128
Beans, baked (home).....	3 hp. tbsp.	150	298
Beets.....	2 hp. tbsp.	70	29
Carrots.....	3 hp. tbsp.	100	18
Celery, creamed.....	3 hp. tbsp.	90	66
Peas, green.....	3 hp. tbsp.	92	110
Potato, baked.....	med. size	130	149
Potato, mashed.....	2 hp. tbsp.	100	112
Spinach.....	2 hp. tbsp.	100	57
<i>Cereals.</i>			
Bread, white (home).....	1 slice ($3 \times 4 \times \frac{1}{2}$)	37	100
Bread, white (baker's).....	1 slice ($3\frac{1}{2} \times 3 \times \frac{1}{2}$)	30	80
Bread, whole wheat.....	1 slice ($3\frac{1}{2} \times 3\frac{1}{2} \times \frac{1}{2}$)	42	106
Bread, toasted.....	$\frac{1}{2}$ slice ($4 \times 2 \times \frac{1}{4}$)	10	31
Zwieback.....	1 slice ($3\frac{1}{2} \times 2 \times \frac{1}{2}$)	15	65
Farina.....	2 hp. tbsp.	100	56
Hominy, boiled.....	2 hp. tbsp.	100	84
Oatmeal, boiled.....	2 hp. tbsp.	100	63
Rice, boiled.....	1 hp. tbsp.	100	112
Macaroni, boiled.....	2 hp. tbsp.	100	91
Corn-meal mush.....	3 hp. tbsp.	115	96
<i>Miscellaneous.</i>			
Eggs, boiled.....	1	50	83
Omelette.....		75	177
Apple, baked.....	1 large	120	128
Apple-sauce.....	3 hp. tbsp.	125	201
Prunes, stewed.....	4 large, with juice	200	189
Cocoa.....	1 cup	227	279

CHAPTER VIII

DIET IN SICKNESS

Only general rules can be given here, in addition to, or by way of summary of, what has been said in various places under the different headings of infant-feeding, and of what will be included in the study of the individual diseases.

In the case of breast-fed infants under 1 year, the development of acute gastric disturbance often renders it advisable to stop nursing entirely for 12 or more hours, giving barley-water or other cereal decoction in its place. The mother's milk may be examined and, if faulty, modified if possible. The withdrawal of the breast may continue for a few days if necessary, but be made permanent only if symptoms of indigestion are persistent or repeated and the infant ceases to thrive in other respects. Mixed feedings should be tried before weaning the child completely.

In the case of bottle-fed infants acutely ill with some digestive disturbance, a general rule which can be wisely followed in most instances is to administer a purgative and to stop the milk-mixture entirely for at least 24 hours, giving a weak cereal decoction, such as barley-water, in its place. After this a graduated but fairly rapid return to the former food may be made. It is a mistake to institute any permanent radical change in a diet which has previously agreed well. Should, however, the digestive disturbance constantly recur, and the infant cease to do well in other respects, being a case of "feeble digestion," a very decided alteration of the diet or of other factors connected with it should be made promptly, since the longer the disturbance continues, the greater the effect upon the infant, and the harder it is to restore it to a condition of health.

What change shall be made can be determined only by a careful study of the individual case. When persistent *vomiting* is the symptom present, it is oftenest the fat which is at fault, and the percentage of this must be reduced very greatly. Food in which skimmed-milk is the basis is frequently very useful in this condition. If reduction of the fat does not succeed, the sugar should be suspected. Sometimes the employment of lactose instead of maltose or saccharose is efficacious; sometimes, contrary to what is to be expected, the reverse is the case; sometimes no sugar is well borne. Buttermilk has the advantage that it is low both in fat and sugar, and is sometimes very serviceable. Even when fortified with wheat-flour and cane-sugar, it is still of value in many cases of vomiting, provided this is not dependent upon a sugar-intolerance. In this connection we must not forget that it is not alone the nature of the nourishment but other factors which may cause and maintain vomiting. Often the food is given in too large an amount, too frequently, or too rapidly. Whether it is better to give a large quantity of a diluted food or a smaller amount of a more concentrated nourishment is a question to be settled often only by trial. Certainly the intervals of feeding should as a rule be lengthened, and the possible existence of other forms of faulty management sought for. (See Action of Milk-elements, p. 127 and Vomiting, p. 700.)

When *diarrhea* is the prominent symptom, again the fat is first to be suspected, especially if the stools contain large numbers of soft, white curds; or the sugar if the stools are green or yellow-green and have a sour odor. Should the infant be thriving, the condition not acute, and the stools not very frequent in number, no immediate change need necessarily be made, but the condition carefully watched. Diarrheal stools if of a foaming character with irritation of the buttocks depend upon an excess of sugar. As lactose and dextrin-maltose, if in large amount, are liable to produce this disturbance, saccharose may be substituted; but it is better to begin with no addition of sugar, and to make this very gradual. The employment of malt-soup, modified in ways to diminish the amount of converted starch present, gives good results in some instances, but in others increases the diarrhea. Buttermilk-mixture (p. 148) is also useful in many instances, and in others casein milk (p. 148); both having a low percentage of lactose, and a high percentage of casein, the latter a moderate amount of fat and the former replacing this by a high carbohydrate-addition in the form of saccharose, which may be tolerated when lactose is not. The casein-milk, at first without sugar addition, is to be selected when it is thought that the sugar disagrees; the other when the fat is especially suspected.

Constipation may call for a modification of the diet. (See also p. 757.) Apart from other causes it may depend upon too small an amount of food ingested as a whole, or of one of the elements, usually fat or carbohydrate. Constipated, thriving children may have such complete absorption from the intestine that little waste remains. When the stools are dry and light colored and perhaps offensive, there is generally an excess of fat and sometimes of protein or a lack of sufficient carbohydrate. The increase of the latter will often change the character of the stools and relieve constipation, and especially so if a combination of starch with a dextrin-maltose preparation be employed, as exists in malt-soup.

The significance of *colic* is very uncertain. Sometimes it depends in no way upon the character of the food, and it is a common attendant upon constipation. When the food is without doubt the cause, the exact method of production of the colic is still frequently uncertain. An excessive carbohydrate-intake, especially of starch, is often a factor, as is also sometimes too high a percentage of protein. This protein indigestion occurs in probably but a relatively small number of cases. The symptoms are indefinite and not entirely understood, the most suggestive in addition to the colic being the occurrence of offensive stools with a putrefactive odor, either diarrheal or constipated, and sometimes of hard, yellow-white protein curds in the passages if the milk has been given raw. In the way of dietetic modification, the casein may be reduced by the employment of whey-mixtures with cream, or the milk may be boiled or be peptonized. (See also *Colic*, p. 728.)

The employment of thin cereal waters in the case of sick children has already been referred to. In some instances, especially in infants past the age of 6 months, benefit in digestive disorders is obtained by giving a stronger cereal-food, entirely without milk at first. This is sometimes digested much better than either fat or sugar. Small and gradually increasing amounts of milk should be added as soon as possible, inasmuch as the diet is generally too insufficient in protein for employment for any considerable time. One of the most trying evidences of malassimilation of the food is that in which, without sufficient vomiting or diarrhea to account for it, there is a persistent failure to gain weight, or even a loss

of it. This may depend upon an insufficient amount of nourishment, but oftener is the final result of the giving of food which was too strong, especially in fat. The employment of a high carbohydrate-diet with a low fat-percentage is at times one of the best remedies. Where a mere insufficient amount of food offered is the cause, the condition is readily curable. In other instances the appetite is very poor, and food is refused. Here good may come from lengthening the feeding-intervals. The feeding of these cases of disturbed digestion will be studied more in detail in considering the diseased conditions in which the symptoms occur.

In children over 1 year of age the presence of persistent weakness of digestion may necessitate the continuance of the use of milk in modifications which would suit younger normal infants. In other cases the best course is to eliminate milk more or less completely for a time. The giving of beef-juice and of scraped underdone meat is often useful under such conditions, while the effects of fat and of carbohydrate, especially starch, must be watched carefully. Each case is a rule to itself, and the diet must be studied and altered as with infants under 1 year. Particularly about the age of 2 years or later there is liable to develop a form of chronic indigestion which depends upon too free a use of amylaceous food. This starchy indigestion requires a modification of the diet by which milk and meat, and, to some extent, green vegetables constitute the principal articles of food, and starch is reduced to a minimum. In my own experience an excess of protein in the diet after the 2d year is less often harmful than is an excess of carbohydrate.

In many of the febrile diseases in infants or older children, vomiting is the first symptom. When this is active, little or no effort should be made to give nourishment. So, too, the dislike for food often seen in acute febrile disorders need be no cause of alarm. It is a natural result of the impaired digestive power always present, and nourishment should not be urged. If the attack, however, is prolonged, or when the condition is such that inanition is threatening from lack of food, the physician must feed sufficiently, although cautiously. It may even be necessary to employ gavage. Curiously, in some cases of obstinate vomiting in infants, food given by gavage, immediately following lavage, will be retained when swallowed nourishment is not. (See Gavage, p. 247.) It is seldom that a sick child may not have water as often and as much as it desires. It is chiefly when the administration of water appears to excite vomiting that the amount given must be restricted. Thirst is very intense in the febrile disorders, and it has been the misguided practice of many of the laity to limit the amount of water. This is both dangerous and cruel. The water should be cool, but not cold. Sometimes, for older children, a carbonated water is to be preferred. Nursing infants with fever should have water given from a spoon, since, if allowed to quench their thirst by nursing, they may readily over-feed themselves. On the other hand, it sometimes happens, where the giving of nourishment is important, that the existence of thirst may be taken advantage of in older children, since, in order to quench it, the child may be induced to drink cool milk if water is withheld; or water may be given as a reward after the milk is taken.

Great regularity should exist in the feeding of sick children. Liquid nourishment should be given every 3 hours, or sometimes every 1 or 2 hours or oftener when very little is taken at a time; but as a rule the interval should be long. The impairment of the digestive power present in the acute febrile disorders, not themselves of digestive origin, often

makes it advisable that the food be weaker than that used in health, and especially that the fat be reduced. Infants who have been receiving a modified milk mixture should have this weakened, and children who have been on solid food require a liquid diet. Of all forms of liquid diet for older children that oftenest serviceable is milk, which should be diluted and may often be alkalized with lime-water with advantage. In the cases where milk is disagreeable to or disagrees with the child we may fall back upon such foods as egg-water or raw egg given in other ways, beef-juice, commercial peptonized beef-preparations, and broths thickened with starchy food. Clear broths, though appetizing, have little nutritive power. Jellies do not nourish to any extent, but well-made ice-cream is often a valuable food in selected cases for children who have lost appetite and need feeding. It should be given, however, rather as a reward, and not made a constant article of diet, as its sweetness is liable to cause or increase indigestion.

In long-continued illnesses often one of the greatest problems is to maintain the state of the general nutrition in a satisfactory manner. In giving, then, the restricted diet referred to, the greatest caution must be exercised not to overdo the matter. I have repeatedly seen children whose chief ailment after some illness was that they had been, and still were, greatly underfed. In cases of obstinate vomiting food may sometimes be given in the form of nutrient suppositories, or, oftener, of nutrient enemata. These, however, do not fill the place which they do in adult life, owing to the difficulty, especially in infancy, of having them retained and the deficient absorption which occurs. As a rule, they are unsatisfactory at this period of life.

CHAPTER IX

CHARACTERISTICS OF DISEASE IN INFANCY AND CHILDHOOD

The diseases of early life vary decidedly from those occurring later. Not only are the causes often different, but the reaction of the growing tissues in early years is not the same as in adult life. Anatomical and physiological distinctions also exist. There is consequently seen a tendency to the development of certain diseases in infancy and childhood, and an immunity toward others. Certain maladies are peculiar to early life; others exhibit clinical manifestations characteristic of that period. The susceptibility of the incompletely developed nervous system is very great, often masking the real nature of the disorder. Trifling factors thus produce general symptoms which are, or appear to be, severe out of all proportion to their causes, similar agencies acting in adults giving rise to no symptoms of moment. The initial effect of deleterious influences is often unusually marked in early life, and the development of symptoms very rapid and apparently severe; while, on the other hand, the recuperative power is great, and convalescence is speedy. Various causes render the examination of a sick child difficult. There are also marked peculiarities at this period in the reaction of the system to certain drugs, some of the *materia medica* being unusually well tolerated, and others not at all so.

After early childhood is passed, and particularly after the age of 7 or 8 years, the peculiarities attending the study of disease are less marked.

ETIOLOGY

Among the causes predisposing to the development of certain disorders in children direct or indirect *inheritance* plays an important rôle. Syphilis in either parent is directly inherited, as probably are very occasionally such of the acute infectious diseases as typhoid fever, scarlatina, variola and some others. Tuberculosis existing in the parents certainly predisposes to its development in the offspring, as do various nervous disorders, such as epilepsy, insanity, the neuropathic diathesis, and some of the muscular dystrophies. Rheumatism and gout are also to be named. Some of these diseases may not actually show themselves until childhood is past, although the seeds of them are present in the system.

To be mentioned also is the etiological influence upon the diseases of the infant of the various *morbid conditions incident to fetal life and to birth*, which will be referred to again (p. 209). Maternal impressions have frequently been considered a powerful factor in the production of some of these; but although much evidence has been adduced in favor of this view, the element of coincidence is too often ignored, and positive proof of any casual relationship is entirely unsatisfactory.

Imperfect feeding and hygiene are among the most active causes of disease in infancy and childhood. As a result arise the many forms of disturbed digestion and their consequences; such constitutional conditions

as rickets and scurvy; the disordered states of the respiratory apparatus so common in children, and the diseases which depend upon lack of proper care of the nervous system. The influence of *school life* is responsible for many nervous ailments, affections of the eyes, deformity of the spine, and disorders of the general health.

Infection, finally, has unusual etiological power in children, the great majority of cases of acute infectious diseases being witnessed at this period. This is partly due to a greater degree of susceptibility; partly to a much greater opportunity of exposure; and partly to the fact that most adults have already become immune through earlier occurrence of these affections.

GENERAL METHODS OF EXAMINATION AND DIAGNOSIS

There are a few respects in which the study of disease in infancy and childhood is easier than in adult life. Existence having been of shorter duration, there is generally a shorter history to be obtained. There are fewer complicating diseases, at least in the acute affections, and there are fewer previous varied bad habits of living to be taken into account.

On the whole, however, the subject presents many and varied difficulties. Many affections exhibit in childhood symptoms different from those of adult life, and the significance of even the same symptom often differs; while there are also many diseases which are almost or entirely peculiar to early life. Again, the infant cannot, without speech, describe its symptoms, and the older child cannot be trusted to give an accurate account. The inability to control the patient presents another difficulty, and the constant crying of an infant may render a satisfactory examination impossible. Considerable skill, patience and tact are therefore required.

Obtaining the History.—As a sequence to what has been said, dependence must be placed on the gathering of a careful and complete clinical history from the mother or nurse. In infancy there is, of course, no history obtainable from the patient, and even up to the age of 6 or 7 years, the child either refuses to answer questions on account of timidity, or makes replies which are not dependable. The presence of pain, for instance, may be denied when it really exists, or maintained when not existing, and the localization by the patient is very misleading, pain in the chest being perhaps referred to the throat or abdomen, and so on. There should be some definite order followed in the procuring of the history from the attendant. Considerable latitude is, of course, allowable, dependent upon the nature of the ailment; yet, in general, the first point should be a brief statement of the present complaint limited to a very few words; next, the past clinical history of the patient; third, the detailed account of the present illness, and, last, the family history. To reverse this order renders the asking of numerous useless questions unavoidable.

After eliciting very briefly the chief symptoms from which the child seems to be suffering, it is often well to allow the mother to give as full an account as possible of the case, in her own way and undisturbed by interruptions, unless these become necessary. Ordinarily the history may best be heard in the presence of the child, who, meanwhile, is growing accustomed to the physician's presence, and who, unknown to itself, is at the same time under the quiet observation of the physician's eye. If the mother's mind is distracted by the child's crying, or if the child

is a nervous one who may be injuriously affected by hearing the questioning, the history should be obtained in another room. While, as a rule, the mother's opinions are of little value, her statements of *what she has noticed* must be listened to and weighed with care; since by her constant association with the child, and by her natural anxiety, her powers of observation have sometimes become very keen, and she is rendered peculiarly able to detect even slight changes from the ordinary condition of health. After the mother has finished her account, she should be subjected to careful questioning by the physician, much, or all, of the child's life being passed in review. Leading questions must be avoided as much as possible, for the suggestions which these offer are very liable to lead to erroneous statements.

Previous History.—In the case of infants this should date from birth; and this applies with equal truth to many older subjects. Among the data concerning which information will be sought are the existence of prematurity, of asphyxia following birth, the nature of the labor, the process of dentition, the time when sitting and walking were begun, the birth-weight and the subsequent alterations, the frequency of micturition, the freedom of perspiration, the character of the sleep, the existence of mouth-breathing, the state of the bowels, the condition of general nutrition and the time when any change in this was seen, the condition of the nervous symptoms in general, and the psychic development, often best shown by the degree of interest taken and the ability to understand and to talk. Particular attention must be paid to the minutest details of the different methods of feeding which have been employed and to the effects of these; whether weaning has taken place, and, if so, why. The statement, for instance, that the baby had been fed on modified milk is not sufficient. The *proportions* of milk, water and the like should be ascertained. Especial care must be given, too, to discovering the date, nature, mode of onset and duration of all previous illnesses. This is often a matter of some difficulty, and may require careful, patient questioning.

Present Illness.—We can next take up the illness from which the child is now suffering. The exact date of onset and the sequence of the symptoms is a matter of importance, and may require time and skill to elicit. At least the mother is generally able to say at what time she considered the child to have been entirely well. A detailed description of the symptoms which have been observed is to be sought for. The nature of the questions varies, of course, with the case. The degree and persistence of fever is important, although the history in this respect is of little value unless the thermometer has been used. Interrogations should be made regarding the state of the general nervous system, as, for instance, the occurrence of restlessness, crying, sleeplessness, drowsiness, coughing, twitching, and the existence of pain; the degree of apparent exhaustion or prostration determined by the previous desire of the patient to be in bed or out, and the presence or absence of a desire to play with toys; the position in bed; and the like. In digestive disorders the history should be obtained of the mode of onset and the nature of the food at the time, the history of vomiting or diarrhea and the number of daily occurrences of these, the character of the vomitus or the stools, and the state of the appetite.

Family History.—The nature of the questions asked will naturally depend upon what has already been learned. They should include the clinical history of other children of the family, the parents, and often

of the grandparents and other direct or indirect antecedents when the existence of a possible inheritance is involved. Among the matters which need to be investigated are the general health of the family and antecedents, the occurrence in them of tuberculosis, nervous disorders, rheumatism, gout, insanity, alcoholism, the number of children living or dead, and the causes of death. The existence of parental syphilis must usually be approached with caution. In place of direct questions information may be gained, for instance, regarding the occurrence of numerous miscarriages, the history of maternal cutaneous eruptions, and the like.

Method of Examining the Child.—Now follows the direct examination of the child—an examination which, as stated, has been all this while quietly going on to a certain extent. Very young infants take no notice of the physician's presence. Older children, however, often have much fear of strangers, and perhaps, from previous experience or suggestion, especially of physicians. A quick glance at the patient on entering the room, or the manner in which a word of cheery greeting is received, will often reveal something of the peculiarities of the child in this respect. It is often a good plan to seem to ignore absolutely even the existence of a somewhat timid or irritable child. It quite frequently happens that the confidence which this inspires, as well as a certain degree of pique which it occasions, will soon cause the child itself to make advances toward a further acquaintance; whereupon a skilful response to its overtures will soon establish the most friendly relations between doctor and patient. Avoidance of all hurry, and the use of gentle words and actions, often aid in rendering a child willing to submit quietly to an examination. Sometimes this can be made to appear a game which the physician is playing with his little patient.

In the case of many children whose timidity seems too great to be overcome, and especially with those who have been spoiled by indulgent parents, and who seem to resent in an ugly spirit the physician's presence, nothing whatever is gained by delay, and it is best to proceed quietly, gently, yet firmly with the examination, regardless of any objections made. Many such children, observing that the physician goes on with his work in spite of protest, learn the uselessness of this and behave better at future visits. Very often the difficulty is with the mother rather than with the child.

A sincere love for children, a quick recognition of a child's peculiarities of disposition, a ready adaptability to meet them, and above all, continued experience rapidly lessen the difficulties in the study of disease in early life. Fortunately, practice enables a physician to make an examination of many unruly children almost as satisfactorily as of those who are quiet and docile.

Order of Procedure.—The order of procedure in making the physical examination depends somewhat on circumstances, and the greatest flexibility in the plan is to be allowed. Those examinations are first made either which arouse least objection or which are most important. If the child is sleeping, or often if it is lying awake in its bed, a superficial inspection of it may be made. Under the same circumstances, the time is favorable for determining the character of the radial pulse and for the palpation of the abdomen, for should the infant begin to cry, satisfactory examination of the abdomen is rarely possible. Next, the temperature should be taken before its elevation can have been increased by prolonged crying. Following this, the general inspection should be

completed, the child being undressed for this. Should crying or coughing occur the character of these may be noted.

The physical examination of the thorax may next be carried on, auscultation being practised first, if the child is quiet, since this often causes less alarm and less tendency to cry than percussion does. At sometime during quiet the reflexes may be tested. Last of all the mouth and throat must be inspected and often the nose, eyes, and ears. The urine should be obtained for examination and in many instances the study is not complete without an examination of the blood and of the cavities of the thorax, abdomen, or spinal canal for the presence of fluid and the character of this. Special cases require a radiological and bacteriological study.

Some of these methods will now receive consideration in fuller detail. The clinical significance of the observations made will be considered in the chapter upon Symptomatology (p. 194).

Inspection.—This furnishes often greater results than any other method for arriving at a diagnosis. It is the first examination to be made, and begins, although at a distance, as soon as the physician comes into the presence of the patient. After a general inspection made of the child, while asleep or awake, it should, when possible, be undressed, wrapped in a warm blanket and laid on the mother's lap or on the bed. When a condition of exhaustion, low bodily temperature, the presence of pain on movement, the existence of diseases of the upper respiratory tract, or other cause renders complete undressing undesirable, the body must be examined part by part throughout before the study is ended. Unless the child is thus undressed very valuable data would be necessarily undiscovered; such as differences in the form and degree of motion of the two sides of the chest; the position of a visible apex-beat; the presence of epigastric episternal pulsation or retraction; the occurrence of important eruptions of the skin; the distention of the abdomen or of the abdominal veins; visible peristalsis; beading of the ribs; etc. In addition to these matters inspection must take cognizance of the state of the general nutrition; the physiognomy; the color of the face and lips; the shape of the head; the condition of the fontanelle and of the eyes; the shape of the abdomen; visible glandular enlargement; the position of the body in bed; the shape and movements of the limbs; the condition of the genitals; the character of the sleep; the degree of restlessness or of extreme quiet; the method of nursing; the existence of nasal or oral discharge; the character of the cough and cry and of the stools, urine and vomited matter. The rate and rhythm of the respiration is to be determined; the occurrence of mouth breathing noted, as well as the existence of bronchial rattling which can be heard at a distance, and the presence of dyspnea and its degree and method of manifestation is to be studied. The character of respiration and its regularity may be observed well by watching the moving of the thorax and abdomen while the child is asleep. Nothing can be determined if the child is at all excited.

Special inspection of the *ears* is often required, and should be practised in all doubtful cases where there is unexplained fever, pain, restlessness or stupor. Examination of the *eyes* often gives valuable information. The presence of conjunctivitis is to be noted and of swelling and discoloration of the lids. The use of the ophthalmoscope is possible even in very young children, and important conditions of the eye-ground may often be discovered. Inspection of the *nose* with the mirror and speculum can be carried out in docile children. That of the anterior nares is im-

portant and unattended by difficulty. It may reveal the presence of nasal diphtheria, so often unsuspected.

Inspection of the *mouth* and *throat* should always be made as a matter of routine at the first visit, and the possibility of subsequent development of disease here always kept in mind. The condition of the tongue, gums, and teeth can often be seen if the patient is crying. For a more thorough inspection of the mouth in infancy, the fingers, previously well washed, may be pushed gently in between the gums at one or both sides of the mouth. This, or the gently pressing of the chin downward will be enough to open the mouth, and to allow a visual examination of it to be made easily. To view the pharynx satisfactorily demands quickness and a certain degree of dexterity. A tongue-depressor is required, and for this nothing answers better in family practice than a teaspoon with a smooth handle free from sharp irregularities. Serviceable for office use are the wooden depressors (Fig. 26*b*) made for this purpose, and in occasional cases, where considerable force is required, the excellent rigid depressor devised by H. D. Chapin (Fig. 26*a*).

The infant or young child should be seated in the mother's lap, facing a window. Its hands should be held, or better still, a blanket or shawl be wrapped about the arms and body close under the chin. All preparations are to be made before the physician approaches the child. In this way much less fright is occasioned. He now stands slightly to one side and in front, places one hand upon the head in order that he may steady it and turn it in any direction desired, and with the other hand introduces the depressor, utilizing a moment when the child opens its mouth to cry or to make some remonstrance. With older children who have learned to keep the mouth tightly shut, the spoon can readily be worked in from the side, or the nostrils compressed for a moment to make the mouth open. Every movement must be gentle and without hurry, yet quickly carried out. As soon as the depressor is in the mouth, it is pushed gently backward. When it reaches the base of the tongue the child gags and necessarily opens its mouth widely, the soft palate rising, and the tongue sinking. At this moment, a rapid yet complete inspection of the fauces can be made. All this procedure may be accomplished in a few seconds. The only exception to this is in the case of young infants. Here the reflex gagging is less well-developed, and forcible depression of the tongue is required. It is in such cases that the Chapin depressor is serviceable.

In the case of older, vigorous, and very obstreperous children who struggle violently, a somewhat different plan of holding is required. The mother seats herself facing the light, with the child in her lap and its back against her body. Wrapping her knees about its feet and legs she grasps its left hand in her right and its right in her left and draws them toward her. This movement crosses the child's arms over its chest and draws its head against the mother's breast, thus rendering the patient powerless. When the physician wishes to have both of his hands

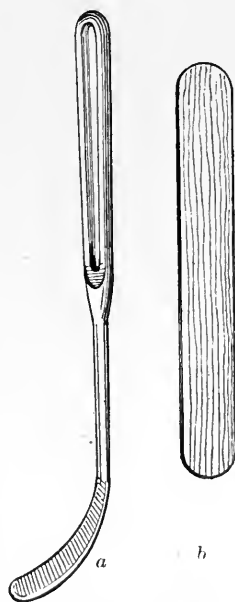


FIG. 26.—TONGUE DEPRESSORS.

(a) Chapin's; (b) Wooden.

free for the treatment of the throat, a modification of this method can be adopted, as shown in the illustration (Fig. 27).

Occasionally timid little children, who have learned to fear a depressor will tolerate the physician's little finger pressed for a moment against the base of the tongue. The finger must, of course, be well cleansed immediately before its insertion. The possibility of having the finger bitten must not be forgotten. Some children will themselves depress the tongue with their own finger. Under certain circumstances, as, for instance, in cases of extreme weakness, it is often best to omit the examination of the throat unless it cannot be foregone.



FIG. 27.—METHOD OF CONFINING THE ARMS AND LEGS TO PERMIT OF EXAMINATION OF THE THROAT.

(Freeman, *Philadelphia Polyclinic*, 1895, March 23.)

The inspection of the *larynx* in infancy and childhood is often important. It is, however, generally difficult to accomplish satisfactorily in young subjects, even by skilled observers.

Palpation.—In palpating a young child it is important to avoid causing alarm. The hand of the physician should be warm and every touch light. When the child is asleep it is well to attempt to take the pulse or to palpate the abdomen. To examine the pulse of the sleeping child the warm fingers may be applied very lightly over the radial artery, and the physician's hand should readily follow, without restraining, any movements of the hand of the patient. If the child is awake its attention may often be diverted by toys. If it becomes alarmed observations of the pulse are rendered worthless. In infants under 6 months, it is often impossible to feel the radial pulse. In such cases it may be counted at the fontanelle. With similar precautions the rate of respiration may be determined by placing the hand upon the abdomen.

To palpate the abdomen satisfactorily it is necessary that its walls be relaxed. During sleep, or when the child is being diverted in some way, is the time to be preferred. The flat of the warm hand should be laid gently upon the skin under the clothing or blanket. Light palpation is employed first, in order not to awaken resistance of the abdominal walls. Local tenderness may be discovered and the degree of any unusual resistance may be gauged. The edge of the liver or of an enlarged spleen may be detected, and any tumors superficially lying can often be readily felt. Now deeper palpation may be attempted. If this causes resistance and the child cries the hand may sink a little deeper every time a breath is taken, and, by maintaining the advantage thus gained, even conditions deep-seated within the abdomen may sometimes be detected. The value of bimanual palpation is not to be overlooked. Sometimes rectal examination may aid greatly a simultaneous palpation of the abdomen with the other hand.

It is often better to do this under anesthesia. When a child continues to scream violently and continuously, abdominal palpation is generally unsatisfactory. At times by keeping the hand in position, and diverting the child, an opportunity to examine may after all be obtained.

In palpating the chest, one seeks for the position of the apex beat, differences in the expansion of portions of the thorax, the presence of the rachitic rosary or other bony alterations, bulging or retraction of any region, the presence of precordial thrill, and the fremitus communicated by bronchial ronchi and less frequently by the voice. Sometimes bimanual palpation in an intercostal space may detect fluctuation in cases of pleural effusion. Palpation is a most important means of diagnosis in cases of retro-pharyngeal abscess as also of adenoid vegetations in little children. In either condition the finger introduced into the nasopharynx will render the diagnosis easy.

Percussion.—This must always be lightly performed. Not only is heavy percussion a cause of discomfort, but it defeats its own object. The chest-walls of the child are so thin that a heavy blow causes a general reverberation and conceals the dullness of a small spot which would otherwise have become apparent. The finger used as a pleximeter should be firmly applied, but the percussion should be done gently and with but one finger, and with a movement of the finger only or the slightest action of the wrist. The feeling of the resistance is as important as the hearing of dullness present. Crying causes no real difficulty, as the tapping can be done at the moments the child stops to draw breath. When there is no crying, percussion should always be made during both inspiration and expiration. In percussing the back or the axillæ, the child may sit in the lap or in the bed; or, in the case of infants, may be held upright in the nurse's arms with its head looking over her shoulder. In either case, however, it is very important to see that its position is straight; not with one arm greatly elevated as compared with the other, or with the spine at all twisted. A faulty position will give untrustworthy results. Indeed, a chest much deformed from spinal disease often cannot be percussed with satisfactory results. A child too ill to be kept in a sitting position may be placed flat on its abdomen. This position may sometimes conveniently be made use of in less feeble patients, since it is one to which healthy infants are accustomed in being dressed. For the examination of the front of the chest the child may conveniently be laid on its back in bed. Percussion should not be too long continued, in order that too great annoyance or fatigue may not result.

Percussion is not so satisfactory in children as in adults, owing to the smaller and more resilient chest and to the variety of sounds produced. It is extremely easy entirely to overlook small areas of consolidation. Even considerable pleural effusion may give no very dull sound. On the other hand, it is not uncommon to discover areas of apparent dullness with which there are neither symptoms nor other physical signs to correspond, and which have no pathological significance, as far as can be discovered. This is especially true for the apices, as has been pointed out clearly by Hamill¹ and by Mielke.² Among other peculiarities of percussion in healthy children is the loud, full note obtained over the whole chest, which would be called tympanitic in adults. A cracked-pot sound is often easily obtainable even in perfectly healthy children who are crying, or even who are entirely quiet. (See also p. 199.)

¹ Arch. of Ped., 1907, XXIV, 92.

² Berl. klin. Woch., 1914, LI, 1218.

These difficulties and causes of uncertainty in no way militate against the importance of a careful percussion of every chest in which disease is suspected, since this examination, combined with experience in disease in children, will often be of the greatest possible service.

Percussion of the abdomen reveals the presence of unusual gaseous distention, free fluid, enlargements of organs, morbid growths, or inflammatory processes (p. 200).

Auscultation.—Auscultation of the lungs in children, and especially in infants, may well be done with the ear applied next to the chest or with only a thin unstarched garment between. This method is suitable when we are seeking for the presence of the scattered coarse râles of a bronchitis, or for large areas of bronchial or of feeble breathing; but for the more exact localization of sounds, or the discovery of lesions in doubtful cases, a stethoscope is indispensable. Yet both the mediate and the immediate method should always be followed, for it sometimes happens that sounds undiscovered by the one will be revealed by the other. Either the disc, double stethoscope or that with the ordinary bell-piece may be employed, according to the preference of the examiner. Children often dislike greatly not only the appearance of the instrument, but also the sensation of the hard cold bell-piece pressed against the skin. The first difficulty can be overcome by allowing the child to handle or play with the stethoscope before it is used; the second by warming the part which is to touch the chest, or by having it covered with a soft rubber ring or surrounded by a rubber cup. The bell must be small in order to make it adapt itself well to the surface of a child at all thin. The use of the soft rubber referred to is an aid to this. The whole chest-piece ought to be short in order to make shifting of the stethoscope easier. The posterior part of the chest should always be auscultated first, not only because it is the more fruitful in auscultatory signs, but because it may be examined more quietly before the child discovers what is going on. Yet all parts must be carefully studied, never forgetting the axillæ. The child may be seated or held in the arms for the examination of the back. Infants may sometimes lie on the abdomen upon the mother's knees. When the child is too ill to sit up, it may be made to lie on the abdomen, or first on one side and then on the other. This last method is, however, less satisfactory, as it prevents immediate comparison of regions on opposite sides of the chest. The lateral portions are best examined while the patient is sitting or held; the front while it is lying in bed. The child must never be so low that the position of the physician is made uncomfortable through stooping, as otherwise the congestion produced interferes with exact hearing and accurate results cannot be obtained.

Crying does not interfere materially with auscultation of the lungs, if the ear is at all practised. Inspiration can be listened for when the child draws breath between the cries, unless there is noisy laryngeal inspiration. In fact, crying is often advantageous in making inspiration deeper, and it is frequently only during crying that vocal resonance can be properly studied in infancy. Increase of vocal resonance may sometimes be determined by observing the closeness of the râles to the ear in a certain locality. Auscultation of the heart-sounds is interfered with considerably when the child cries. They can then be heard only when the child inspires. The irregularity in respiration, especially present in infants, is to be borne in mind, as the very long pauses which occur might deceive the unwary into believing that respiration was inaudible.

Among some of the peculiarities of auscultation in childhood, the

loudness and harshness of the respiratory murmur is to be mentioned, a condition which when present in adults is denominated "puerile" and is considered pathological. In infancy, however, respiration is superficial and feeble. The ease with which the heart-sounds can be heard at the back, and in fact all over the thorax, even when there is no consolidated pulmonary tissue intervening, and with which râles and even bronchial respiration produced in one lung can sometimes be heard in the other is to be noted, as is also the facile transmission through the lungs, especially to the apices, of sounds produced in the upper respiratory tract. It is especially, too, in children that respiration approaching a bronchial character is heard under the clavicles and in the interscapular spaces close to the spine, particularly on the right side, yet without pathological significance.

The results of auscultation in pneumonia in early life often present important differences from those obtained in adults. This is so true that the diagnosis of this disease must often rest upon the symptoms rather than physical signs. The lateness of the development of physical signs is often characteristic, for the attack may sometimes nearly run its course before any signs whatever can be detected. This is perhaps especially true in bronchopneumonia, in which disease the smallness of scattered patches may produce no characteristic alteration of the respiratory murmur. (See p. 199.)

Temperature-taking.—The effort to determine the existence or non-existence of fever by applying the hand to the skin is entirely too untrustworthy. High temperature, it is true, can often be detected if the hand is placed under the clothing upon covered parts of the body, but the method has repeatedly led to grave mistakes. The use of the clinical thermometer is the only means to be relied upon. For employment in children it is much better to have one which will record *quickly*. The one-minute, or still better the half-minute, thermometer is to be preferred, allowing it to remain in place a trifle longer in order to insure a reasonably accurate record. The only absolutely accurate method consists in leaving the instrument in position until the maximum temperature is reached, watching the mercury meanwhile to determine this. The dislike of most small children to temperature-taking is, however, too great to permit of employing this plan. A little experimenting with a quick self-registering thermometer will show how long it generally requires to reach the maximum with approximate correctness.

In well-trained children of 4 years the thermometer may be placed in the mouth, although there is even then some danger of having it bitten. In younger subjects the rectum is the only proper place. The use in the axilla requires very variable times and gives varying results, owing to the difficulty in getting close apposition of the opposing cutaneous surfaces. This is especially true of wasted children. Moreover, this locality requires that the arm be held firmly, and to this children strongly object. It must be remembered that the temperature in the mouth or rectum during fever is at least a degree higher than that in the axilla, even when the latter is accurately ascertained. The employment of the groin or of the popliteal space is to be condemned. The results are seldom accurate.

The bulb of the thermometer should be slightly oiled and pushed into the rectum until well out of sight, the infant meantime lying in any position comfortable to it. The nurse should then keep the tip of her finger upon the end of the instrument. There is no necessity of restraining the

infant's legs, and it may be allowed without danger to kick all it desires if the hand merely guards the thermometer. It is better that the rectum be empty of feces, but this is not an essential. Only when the thermometer in the rectum produces pain and straining, as in some cases of diarrheal disturbance, should the axilla be used for obtaining the record. The time for taking the temperature should not be after a hard attack of crying, as the height of it may be increased. For the same reason, crying during the observation should be prevented, if possible, by diverting the child.

Examination of the Urine.—The obtaining of the urine is often a matter of some little difficulty in infancy. In the case of male infants it may be procured by applying a condom over the penis, or a small bottle with a sufficiently wide neck which may be held in place by bandages or adhesive strips extending to the waist. For female infants the neck of the bottle may be passed through an oblong piece of adhesive plaster, firmly attached there in a way to prevent leaking, and the plaster then applied over the vulva and perineum. In some cases it may suffice to place a large wad of absorbent cotton inside the diaper immediately under the genitals. The child must then be examined frequently in order that the urine may be expressed from the cotton as soon as possible after it has been passed. The method is not very satisfactory. Still another device is to allow the child to lie for a time upon a rubber-cloth and without a diaper, or upon a small circular rubber air-cushion with a hole in the center and a small pus-basin placed under the hole. The surrounding portion of the bed is built up with pillows to the level of the cushion, and the infant, with diaper removed, allowed to lie upon the bed and cushion until the urine is passed into the basin. Children a year old, or sometimes less, may be put at frequent intervals on a chamber in the hope of procuring urine in this way. In the event of none of these methods succeeding, a small silk catheter, No. 9 or 10 French scale or No. 4 American scale, may be employed. Except in very young infants, and sometimes even in these, the instrument can be passed without difficulty. The greatest precautions must be taken against infection, and the method employed only when the obtaining of uncontaminated urine is necessary, as in cases of suspected pyelitis in female infants.

Blood and Blood-pressure.—The examination of the blood is accomplished as in adult life, testing the hemoglobin percentage, the number of and changes in the erythrocytes and leucocytes, and in some cases the coagulability, fragility of the corpuscles, specific gravity, and any chemical alterations which may be present. The blood-pressure, too, is estimated as in adults, with such modifications of the apparatus as the smaller size of the patient often demands.

Puncture of Serous Cavities.—In very many diseases a diagnosis cannot be made without the puncture of the pleural or peritoneal cavity, or of the spinal canal. The procedure is easy and safe if proper precautions are taken. A glass hypodermic syringe with a stout needle is to be employed, or one of the larger syringes made especially for the purpose. Great precautions must be used to disinfect thoroughly the instruments and the skin. The needle and syringe should be thoroughly boiled and the fluid obtained injected into a sterilized test-tube and stoppered with scorched cotton. General anesthesia is seldom necessary, and even local anesthesia is rarely required, the discomfort of the freezing being greater and longer continued than that of the puncture. Fuller details

regarding the employment of puncture will be given later. (See Empyema, Vol. II, p. 113, and Lumbar Puncture, Vol. II, p. 235.)

Radioscopy.—Of recent years the use of the x -ray for purposes of diagnosis is constantly becoming more important. Although originally of principal value in surgery, it has been found serviceable as an aid in medical diagnosis as well. The outlines of the heart, the existence of areas of pneumonia or of tuberculosis, the presence of pleural effusion, the size and position of the liver, and other physiological and pathological conditions may be discovered in this way. A very serviceable purpose of the fluoroscope is the observation of the movements of the heart, lungs and stomach, and, with the aid of bismuth, the rapidity of the emptying of the last-mentioned organ.

Electrical Examination.—This is of great value in many instances of nervous disease for the determination of the extent and nature of paralyses of different sorts, and the reaction characteristic of a spasmophilic state. To obtain the reactions satisfactorily it is sometimes necessary to give an anesthetic.

The Reflexes.—The knee-jerks may be tested when the child is sitting, diverted on its mother's lap. A good plan is to support the foot with one hand. Any jerk which occurs can then be felt as well as seen. Another method consists in grasping the thigh with one hand just above the knee. In this way the contraction of the quadriceps extensor muscle can be felt readily. The knee-jerk cannot always be elicited easily, sometimes owing to the flabbiness and weakness of the muscles, sometimes to the difficulty in obtaining a voluntary relaxation on the part of the child. The existence of ankle-clonus and of the Babinski and other reflexes of the lower extremities should be investigated and sometimes those of other parts of the body as well. The matter will be referred to again in the section upon Nervous Diseases (Vol. II, pp. 233, 322).

CHAPTER IX

SYMPTOMATOLOGY AND DIAGNOSIS

The characteristics of appearance and development present in health have already been considered in discussing Anatomy and Physiology. The symptoms pertaining to diseased states in early life and the significance of these may now be reviewed briefly. A more complete description of many of them will be found under the headings of the individual diseases.

SIGNIFICANCE OF SYMPTOMS

Position and Movements.—A child with a commencing illness which is attended by pain or fever no longer exhibits the quiet, motionless sleep of health, but tosses from side to side. When awake, too, the natural restlessness is in like manner increased, the patient wishing to be taken from bed, put back, rocked, or carried about in many ways, indicating the excited state of its nervous system. On the other hand at the beginning of an infectious disease it often happens that the patient will lie unusually still, sleeping constantly, the evidence apparently of the toxic state existing. All movements are slow in debilitated states, and a child afflicted by very profound exhaustion may lie for hours without motion, with its face directed upward instead of to one side, as it commonly is in health. The same position and lack of motion is seen in coma from any cause.

Restlessness in infancy especially during sleep, with an unusual tendency to kick the covers away, is an early symptom of rickets. Restless sleep may depend upon hunger, pain, nervousness, great fatigue, noises or light in the room, or unusual excitement before going to bed. Later, restlessness at night may accompany certain forms of chronic gastro-enteric indigestion. An intense degree of restlessness, called "jactation," may occur in some respiratory diseases, especially those of the larynx; in a state of acidosis with air-hunger; in severe chorea; great cerebral anemia and sometimes in severe attacks of infectious disease including sepsis.

Orthopnea may attend diseases of the respiratory apparatus or of the heart, the child resting comfortably only when propped up in bed or when held upright in the nurse's arms with its head against her shoulder. Sleeping with the head thrown back and the mouth open is often the result of obstruction to respiration by adenoid growths. A rocking of the head from side to side on the pillow may be observed in infants with rickets, sometimes in meningitis or headache, and is frequently a natural expression of an intensely nervous state. Keeping the head bent backward is seen in basilar meningitis of different forms, and to a less degree in headache. A fixity of the head and neck also attends cervical caries or torticollis, the head in the latter condition being generally turned to one side. Boring the head into the pillow may indicate meningeal disturbance. Lack of power to hold it erect denotes great general weakness, or may be an evidence of congenital or acquired torticollis, of idiocy, or

of some other nervous disorder, such as, in later childhood, advancing Friedreich's ataxia. Inability to walk may be the result of idiocy, of actual paralysis, or of a pseudo-paralysis dependent upon syphilis or, oftener, rachitis. Failure to move one or more limbs properly may denote paralysis, but may equally well be due to the pseudo-paralysis of rickets or syphilis, or depend upon congenital dislocation of the hip-joint, or indicate that motion is avoided because painful, as in infantile scurvy. Lying upon one side sometimes occurs in pleural effusion of that side. Lying with the head retracted, the back hollowed, the knees and elbows flexed and the arms crossed over the chest—the so-called “gun-hammer position” (*en chien de fusil*)—is frequently a symptom of meningitis. Lying upon the abdomen may indicate abdominal pain, but with many children it is only a habit.

Often an infant will repeatedly put the hands fretfully to the head when there is headache, to the mouth when pain exists there, or to the ear when earache is present. In the latter condition the side of the head is frequently held pressed against the pillow or the mother's breast. Pulling at the ear accompanied by fretfulness may, however, be only a nervous habit in a child suffering from rachitis or other debilitating disease. Rubbing or picking at the nose indicates coryza or gastro-intestinal disturbance or is a neurotic habit merely. Pulling at the throat sometimes occurs when there is much dyspnea. The violent alternate flexion and extension of the limbs upon the trunk, and of the trunk itself, accompanied by clenching of the hands and the characteristic cry, denotes the pain of colic. The keeping of the thumbs drawn into the palms and the toes flexed or rigidly extended often indicates impending convulsions, and it is also present in tetany. At the same time it is to be borne in mind that every very young infant has a tendency to keep the thumbs thus inverted much of the time. Rigidity of the limbs may occur, as in meningitis, cerebral paralysis, and spinal caries. More or less extensive tonic spasm may be seen in tetanus. Irregular, jerking, incoördinate movements in older children occur in chorea. Clonic, to-and-fro movements with unconsciousness are characteristic of convulsions, while true ataxic movements are seen in Friedreich's ataxia and allied conditions. Tremor is rarely observed in children, except as a result of chorea, some organic nervous disease, or of such weakness as develops after fever. The shaking of a true rigor is uncommon in early childhood and infancy, being replaced by coldness, pallor, drowsiness, unusual quiet, or a convulsion.

Surface of the Body.—A yellowish tint of the cutaneous surface and of the conjunctivæ is seen in icterus. Flushing of the face is common in fever, and is also often observed in chronic gastro-intestinal indigestion of older children, and from the action of belladonna. Slight eczema or chapping of the cheeks simulates flushing to some extent. A flush which comes and goes slowly, on the face or on a part of it, or on the trunk when exposed to the irritation of the air, is a characteristic symptom frequently present in meningitis. The broad red line which develops after drawing the finger over the abdomen in cases of meningitis (*tâche cérébrale*) is of the same nature. This symptom is, however, not pathognomonic but only suggestive of this disease. Very marked blueness of the whole face, the fingers and toes, and the mucous membrane of the mouth, is present in congenital cardiac affections, less often in the intense dyspnea arising in laryngeal stenosis, and sometimes in severe pneumonia. A slightly bluish tint of the lips and cheeks is of common

occurrence in cases of post-natal affections of the heart. The red flush present on the cheeks in many cases of pneumonia quite commonly has a bluish tint to it. Moderate distention of the veins running over the scalp and at the root of the nose occurs in debilitated children, but especially in rickets. The veins of the scalp are also much distended in hydrocephalus. Great distention of the veins of the face and neck attends any decided degree of dyspnea. Dilatation of the veins over the abdomen and lower part of the thorax is witnessed in cases of malignant abdominal growth or sometimes of tuberculous peritonitis. A faintly purplish tint of and under the eyelids and above the mouth is often seen in infants with debility or even with any slight disturbance of health.

Marked pallor of the skin accompanies nausea, anemia of any sort, rickets, chronic diarrhea, chronic suppurative processes, nephritis, and frequently heart disease. Combined with coldness it may replace in the infant the chill of adult life. An earthy color is frequently observed in severe chronic diarrhea and a brownish-yellow color of the skin, especially of the projecting portions of the face, in congenital syphilis. The various eruptions of the exanthematous fevers are oftenest witnessed in children, since these diseases are far commonest at this age. Infants show an especial tendency to inflammations of the skin, such as miliaria, eczema and forms of erythema. Profuse sweating, especially of the head, is an early symptom of rickets. Coldness of the extremities is present in weakly babies with poor circulation and in infants suffering from colic. Clubbing of the fingers and toes occurs in congenital cardiac disease and in chronic affections of the lungs and pleura. A shining red appearance of the palms and soles in young infants is a symptom of inherited syphilis, while peeling of the skin in older children, seen especially about the fingers, suggests convalescence from scarlet fever.

Edema of the skin, especially of the face and feet, may indicate nephritis or valvular disease of the heart, or may be an evidence of extreme malnutrition and feeble circulation. Angioneurotic edema sometimes occurs in children. A localized asymmetrical atrophy of the muscles points to poliomyelitis or neuritis, while an undue development, especially of the calves, may indicate pseudo-hypertrophic muscular dystrophy. Local swellings of the joints are observed in all forms of arthritis, and the swelling about the joints and of the shafts of the long bones in scurvy is not to be forgotten. Curvature of the spine of various forms may be dependent upon disease of the vertebræ, old pleurisy, unequal length of the limbs, rickets, or faulty positions in being carried or in sitting. General wasting in infancy is oftenest the result of insufficient nourishment, of persistent diarrhea or vomiting or of chronic intestinal indigestion. In some cases, however, it is a sign of tuberculosis or of congenital syphilis.

Face and Expression.—The mouth is open during sleep in cases of adenoid or tonsillar hypertrophy, or when the nose is obstructed by secretion. Chewing movements occur when there is indigestion or inflammation of the mouth. A general puffiness of the whole face, with redness of the eyes, is often present during pertussis or measles. Puffiness is also witnessed in the edema of advanced marantic conditions, and especially about the eyes in nephritis.

Pain is expressed during sleep by contortions of the face of various sorts. Thus there is sometimes an expression of pain with contraction of the brows in headache, while the smiling of very young infants during

sleep often signifies abdominal pain. Discharge from the nose occurs in coryza, and is often, also, one of the first symptoms of nasal diphtheria. The "snuffles" of new-born infants suggest congenital syphilis. In older children persistently reddened eyelids, combined with a swollen upper lip, wide nostrils, thick nasal discharge, muddy complexion, and enlarged cervical lymphatic glands, may indicate the existence of the lymphatic-exudative diathesis with tuberculosis, a combination to which the title "scrofulous" was formerly applied. Wide-open nostrils moving with every inspiration and accompanied often by an anxious expression of face are observed in dyspnea, most commonly from pneumonia or from stenosing affections of the larynx. Occasional movement of the nares is, however, of frequent occurrence in healthy infants. A flattened, somewhat sunken bridge of the nose is characteristic of congenital syphilis, but by itself is not sufficient to warrant this diagnosis. In atrophic conditions in infancy, when the fat has largely disappeared or there has been great loss of fluid through diarrheal discharge, the face becomes lined in a marvellous manner, especially when the child cries, suggesting the features of a very old man. The most prominent of these lines is that called the "nasal," extending from the alæ of the nose and running in a half circle around the corners of the mouth. Other special lines have been described but have little importance.

In severe acute disease, atrophic states, or when there is pain or indigestion, the eyes may be only partially closed during sleep. Twitching of the lids and crossing or rolling upward or outward of the eyes indicates impending convulsions. The eyes in hydrocephalus are directed downward, with the lower part of the iris covered by the lower lid and the sclera above the iris visible. After a severe acute attack of diarrhea or vomiting the tissues about the eyes shrink, leaving them peculiarly large and staring (Fig. 28). The pupils are dilated or unequal, or sometimes contracted in meningitis or other intracranial disorder.

Nystagmus may often be seen under these conditions, but may be a purely functional disturbance combined with spasms nutans or gyrospasm. Strabismus, too, is frequently an attendant of intracranial disease, but is equally well a congenital defect. Keeping the eyes shut or turned from the light or buried in the pillow indicates photophobia from conjunctivitis, keratitis, or headache dependent oftenest on meningitis. A film-like appearance of the cornea develops in children who are moribund. Ulcers on the cornea often occur in syphilitic or tuberculous children. Tubercle of the choroid, choke-dises, and other important interocular conditions may be discovered by ophthalmoscopic examination.

Head and Neck.—Various alterations of the head appear, some of which add to the altered expression of the face. In chronic hydrocephalus the head is globular, the forehead overhangs, and the face looks small. In rickets the head has in general a square or oblong form, with the top flattened and the frontal and parietal eminences unusually large. The face looks small and is given a somewhat square appearance through the widening of the lower jaw. Asymmetrical heads are due to the pres-



FIG. 28.—SUNKEN TISSUE ABOUT THE EYES.

Facies of acute diarrhea and vomiting; girl aged 11 months. (Thomson, *Clinical Examination of Sick Children*, 2d Edition, 15.)

sure by forceps or oftenest to the existence of rickets, the lying too much on one side producing the deformity in the latter case. Faulty position may, however, produce the deformity even when rickets does not exist. Flattening of the occiput may be due to pressure, the result of the rachitic infant lying too constantly upon its back. Asymmetrical, microcephalic, and other deformed states of the head may be found in idiocy. Spots of thin membrane-like bone (craniotabes) may occur in the occipital region of infants in the early months of life. The fontanelle is unduly prominent and tense in hyperemia of the brain from any cause, meningitis, tumor, and chronic hydrocephalus. It is very large with the sutures open in the latter disease, as it is to a less extent in rickets. It is depressed in conditions of inanition or after profuse diarrhea, and especially in collapse. Not infrequently the bones of the skull overlap under these circumstances. The fontanelle closes very early in microcephalus and late in rickets. A systolic murmur is sometimes audible in the neighborhood of the anterior fontanelle, especially in cases of rickets. It may also, however, sometimes be heard in some healthy children. Tenderness over the tragus and over the mastoid may indicate otitis. Swelling of the occipital and superficial cervical glands is often the result of inflammation of the scalp; while that of the glands below the body of the jaw commonly attends affections of the pharynx and nasopharynx. A fluctuating swelling in the neck may be due to abscess of the glands or sometimes to a retropharyngeal abscess pointing here. The hair is worn from the back of the scalp in cases in which there is much rocking of the head, as in rickets.

Mouth and Throat.—Blueness of the lips has already been alluded to (p. 195). Fissuring around the mouth may be a symptom of congenital syphilis. Grinding of the teeth occurs especially in infants with cerebral disease or suffering from convulsions, but it is also heard in children with slight digestive disturbance, and in some cases seems to be only an insignificant although disagreeable habit. Notching of the permanent upper incisor teeth is seen in congenital syphilis. The mucous membrane of the mouth may exhibit mucous patches in syphilis, and is one of the earliest sites for the appearance of the eruptions of some of the infectious fevers. A hemorrhagic swollen condition of the gums is seen in infantile scurvy. The tongue is coated in most disorders of digestion, but in some instances is bright red and smooth. It is coated in many fevers; bright red with prominent papillæ ("strawberry tongue") in scarlatina; worm-eaten in appearance in the so-called "geographical tongue;" cyanotic in congenital heart disease and slightly so in pertussis, and may, like the rest of the mucous membrane of the mouth, exhibit the lesions of some of the forms of stomatitis. In children with severe cough, and especially with pertussis, who have cut the lower incisor teeth, ulceration of the frenulum linguæ is not infrequent.

Cleft palate is a congenital defect, while perforations are generally the result of congenital syphilis. High arching of the palate may attend some forms of idiocy, but may also be present in children with perfect mental condition. It is not infrequently associated with deviation of the septum and the pressure of adenoids.

Thorax.—The chest in rickets is small and exhibits the rachitic rosary in front and bulging of the ribs behind, with marked depression in the lateral regions. On horizontal section this gives the well-known "violin shape." A horizontal depression beneath the nipples is also characteristic of rickets, especially where there has been much disturb-

ance of the respiratory apparatus. A typical pigeon-breast is due often to obstruction to respiration by adenoid growths. This and other very great deformities of the thorax may be the result of curvature of the spine. A very unusual prominence of the precordium occurs in cardiac hypertrophy in heart-disease in children. Diminished expansion of one side with lack of movement of the intercostal spaces is dependent upon pleural effusion of that side, adhesions from a former pleurisy, or, in a lesser degree, upon pneumonic consolidation. Contraction of one side results from old pleural adhesions. Bulging of the intercostal spaces, with lack of movement, occurs in large pleural effusions; yet effusion may sometimes be present and the interspaces still move. Decided dyspnea from any cause produces in children great retraction of the interspaces with each inspiration. The degree of retraction of the epigastrium and all of the lower portion of the thorax which occur, with tugging of the sternocleidomastoid muscles, and sinking of the episternal notch, is often remarkable at this time of life. It is generally greatest in cases of stenosis of the larynx. Yet a considerable amount of moving of the epigastrium combined with lower thoracic retraction is a normal accompaniment of respiration in healthy infants, and still more so in rachitic subjects. Displacement of the heart's apex by pleural effusion is to be noted. Dullness over the manubrium of the sternum or between the scapulæ may indicate the presence of enlarged bronchial glands. In infancy the substernal dullness may be produced by the thymus gland. Other regions of dullness of small size may be occasioned by old pleural thickening, encysted empyema, areas of collapsed lung, or small pneumonic patches. More extensive dullness may be due to pleural effusion, pneumonia, or widespread pulmonary collapse.

An unusually deep tympanitic percussion note, or a Skodaic tympany, may indicate in children the presence of pneumonic consolidation. It may persist throughout nearly the whole attack. A cracked-pot sound is especially often heard in advancing or receding pneumonia, although it is often, too, present in healthy lungs. Localized ringing râles of unusual loudness and nearness to the ear often signify pneumonic consolidation. Feeble respiration over one side of the chest may indicate pulmonary collapse or pleural effusion. It may, however, be the only discoverable evidence of pneumonia in some instances. This has repeatedly led to the erroneous supposition that that side was affected on which the loudest respiration was heard. In other cases of pneumonia a slight harshness of respiration is the only physical sign. The frequency with which numerous râles occur in bronchitis in children as compared with adult life is another interesting feature. Bronchial respiration generally denotes consolidation, but only when accompanied by other symptoms. As already stated it may often be heard through a pleural effusion, and is normally present to a certain extent in certain parts of the lungs. (See p. 190.) In the supraspinous fossæ it may at times be a sign of enlarged bronchial glands.

The various cardiac murmurs heard offer nothing peculiar in childhood to be mentioned in this connection, except that the characteristics of the murmurs of congenital disease of the heart are to be borne in mind, as well as the frequent occurrence of accidental murmurs. These will be referred to in considering Diseases of the Heart (Vol. II, pp. 121, 158). A very distinct precordial thrill occurring in a young infant points strongly to congenital heart disease.

Abdomen.—Unusual gaseous distention of the abdomen is a common symptom of rickets and is also often associated with colic. It is also present in wasting disease due to chronic digestive disorders, is a constant attendant upon idiopathic dilatation of the colon and upon some forms of indigestion in the later years of early childhood and is often a serious symptom in pneumonia. Great flatulent distention with much tenderness may occur in peritonitis, and to a less degree in inflammatory diseases of the intestine. Distention by liquid is noticed in tuberculous peritonitis and in abdominal dropsy from cardiac, renal and, more rarely, hepatic disease. Irregular distention by solid masses occurs in tuberculous peritonitis, fecal accumulation, enlargement of the liver and spleen, morbid growths, intussusception, and in localized inflammatory processes. Marked retraction of the abdomen is seen in cholera infantum, in meningitis, especially of the tuberculous variety and in many exhausting diseases. Absence of movement during respiration may be due to inflammation or to paralysis of the abdominal walls. The outlines of the stomach and of the coils of intestine may often be detected in atrophic children with distention and great thinning of the abdominal walls. Quite active peristalsis is often noticeable in cases of pyloric stenosis and of intestinal obstruction.

Method of Sucking and Swallowing.—Sucking is often almost impossible when the nares are occluded, as by severe acute coryza, congenital syphilis or unusual adenoid growth, since the infant cannot breathe while the mouth is closed on the nipple. Harelip or cleft palate likewise renders sucking difficult or impossible on account of the interference with the production of the necessary vacuum in the oral cavity. Refusal to nurse after making a short effort may indicate soreness of the mouth. In other cases it shows that little or no milk is obtained from the breast. Swallowing with a noisy gulping sound and with a grimace or a cry of pain occurs in soreness of the throat. Sucking for a moment and then stopping to breathe attends pneumonia, while entire refusal to suck may accompany extreme weakness or coma. It is an unfavorable sign. Inability to swallow, even when fed by a dropper or spoon, is seen in tetanus, eclampsia, stricture of the esophagus, and in children extremely ill from exhausting diseases. Choking over the food, with inability to swallow, occurs in severe cases of retropharyngeal abscess. Regurgitation through the nose indicates pharyngeal paralysis, oftenest after diphtheria.

Respiration.—Acceleration of breathing is very common in children, and the rate is out of all proportion to that which similar causes would occasion in adults. It is seen in fever and, very markedly, in pneumonia, and is constantly present to a variable degree in rickets, even when there is no catarrhal disturbance of the respiratory apparatus. The increase in the respiratory rate for each degree of temperature is approximately the same as in adult life, *i.e.*, about $2\frac{1}{2}:1$, with the rate of increase slightly greater than this in infancy and slightly less in later childhood (M. S. Cohen).¹ Any excitement will accelerate respiration greatly in infancy. Dyspnea, *i.e.* labored breathing—which may or may not be rapid as well—may occur in any condition which interferes with proper aeration of the blood. It is most typically seen in stenosis of the larynx from diphtheria, sometimes in retropharyngeal abscess, and in cases of foreign body in the trachea or bronchus. It may occur under other circumstances also, such as pneumonia, pleurisy, diseases of the heart,

¹ Arch. of Ped., 1905, 917.

severe anemia, uremia and acidosis. In moderate dyspnea the inspiration is labored, prolonged and noisy; but in bad cases, the expiration has the same character as well. Sometimes the rhythm of the respiration is altered, and instead of the inspiration being the louder and longer, with the pause following expiration, the latter is the louder and more accentuated and the pause follows a short inspiration. This is oftenest seen in pneumonia. It may, however, sometimes occur in healthy infants if excited. A catch in the respiration is often observed in abdominal or thoracic pain. It is witnessed very characteristically in pneumonia and pleurisy, in which the inspiration is short, and "catchy," and is followed by a moaning expiration, the so-called "expiratory moan." Snoring at night points strongly in children to occlusion of the nasopharynx by adenoid vegetations. A curious spluttering, gurgling respiration is heard in retropharyngeal abscess. The natural great irregularity of the respiration in young children, and especially in infancy, is much increased in cerebral affections. This greater irregularity may also sometimes be seen in painful affections, especially of the chest. Sighing, with unusual intermissions in the respiration, is often present in meningitis, yet it may at times occur in healthy infants. A respiration approaching the Cheyne-Stokes type is frequently observed in early life in disorders of the brain, even though only functional in character. Although commonly of grave import, it is by no means so much so in infancy as in adult life. Great slowing of the respiration may take place in cerebral diseases, as in coma. It also occurs in narcosis from opium. Frequent yawning may indicate serious failure of the circulation or sometimes impending syncope.

Pulse.—The pulse becomes more rapid in febrile conditions and under the slightest excitement. This last is so true that it is almost impossible to come to any conclusions regarding it when an infant is awake unless entire placidity is obtained. Other things being equal, and all undue excitement being removed, the increase for each degree of temperature is not so great as in adult life, and the younger the child, the less is the relative augmentation, the increase for 1 degree Fahrenheit being about 4 beats of the pulse during infancy. This has been pointed out by Cohen.¹ The truth of this statement, which is contrary to the opinion generally held, will be readily admitted on slight consideration. If an infant of a year with a normal pulse rate of 110 to 120 showed an increase of 10 beats for each degree of temperature, as does an adult, an elevation of 105 degrees would produce a pulse rate of 180 to 190, which is clearly more than is usually obtained for fever in otherwise healthy and quiet infants. The relationship of the rate of the pulse to that of the respiration, which is 4 : 1 in adults unaffected with any disease which unduly affects either one or the other, is altered somewhat in infancy, being then about 3 : 1 (Squire).² The natural irregularity of the pulse in young children becomes much intensified in many affections of the brain. In such disorders irregularity is often combined with decided retardation. The pulse is to a less extent irregular in pericarditis and often in chorea. It is unusually rapid in scarlatina, out of all proportion to the severity of the attack, and slower than would be expected in many cases of typhoid fever. It may be slightly retarded in nephritis and the arterial tension is increased. The "trip-hammer" pulse of aortic regurgitation occurs in childhood as in

¹ Archives of Pediatrics, 1905, 915.

² Transac. Obstet. Soc. Lond., 1868, X, 280. Ref., Cohen, *loc. cit.*

adults, and the capillary pulse of this disease may be readily obtained in the finger-nails or in the lips.

Temperature.—Most important in this connection is the observing of the ease with which abnormal alteration of temperature takes place in early life. High elevation may result from slight causes, such as constipation of the bowels or even excitement. Temporary fever is not uncommon in the new born. (See p. 302.) More or less elevation may be seen in children with moderate debility, especially during convalescence from an acute disease. The variations in the course of a febrile temperature, including the difference between morning fall and evening rise, are liable to be greater in children than in adult life. Very high temperature may attend the infectious fevers, some of the cases of milk-poisoning, and the heat-exhaustion occurring in very hot weather. High fever of short duration is borne, as a rule, better in childhood than in adult life.

Depression of temperature is witnessed in such conditions as critical fall, severe diarrheal diseases, collapse, hemorrhage, sclerema neonatorum, congenital heart disease, premature birth, and in very many cases where insufficient nourishment is taken or assimilated. In all weakly children the temperature is readily depressed by external cold.

Cry.—The observation of the cry constitutes one of the most important methods of diagnosis in infants. A healthy, comfortable and contented infant does not cry. A cry of any sort always has a meaning, even though it indicates nothing more than some slight dissatisfaction. Persistent violent crying, rather fretful than sharp, is often due to hunger. It is unappeasable by anything except the giving of food, when it ceases at once and permanently. Sometimes a cry of this nature is in reality dependent on thirst, especially if there has been severe diarrhea. A similarly continuous cry, but more high-pitched and piercing, attends persistent severe pain, most commonly earache. The offering of food quiets it only momentarily if at all. Pain of a less severe nature, the existence of the intense itching of eczema, the pain from the pricking by a pin concealed in the clothing, the presence of a wet diaper, and many other sources of discomfort produce obstinate crying, but of a less piercing and violent character. The cry of colic is very violent, but more or less paroxysmal, a momentary pause being followed by a sudden renewal without discernible reason. It is attended by the movements of the body already described as characteristic of colic (p. 195). The giving of food may quiet it for a time, the warm milk lulling the pain, but it soon returns as bad as before. It may cease suddenly after the expulsion of gas from the stomach or bowel. A similar cry sometimes attends the passage of gravel. A sudden acute pain, such as results from a fall or other slight accident or the touching of some tender part, produces violent but temporary crying, soon appeased. Crying just before, with, or after the evacuation of the bowels indicates intestinal pain, or sometimes pain at the anal opening. Crying may also attend the passage of urine and may denote pain in the bladder or the irritation of scalded areas by the secretion.

A weak, peevish, fretful cry, sometimes almost constant, is heard in many conditions attended by much debility. Under such circumstances speaking to or even looking at the child may start the cry. A louder but fretful cry in a healthy child, often attended by rubbing of the eyes with the fists, indicates sleepiness. An almost inaudible cry occurs in severe stenosis of the larynx and in cases of great exhaustion. Puckering the face into the position for crying but absolutely without sound occurs in

these conditions when extreme, as also after tracheotomy or intubation. The absence of crying is witnessed in comatose states. There is also very little crying accompanying decided dyspnea, such as attends severe pneumonia or pleural effusion, on account of the lack of air for it. The cry of pneumonia is suppressed and short, and the expiratory moan described in considering Respiration is often heard (p. 201). Yet children with pneumonia sometimes cry loudly if the dyspnea is trifling. As a rule, however, loud crying indicates that there is little wrong with the lungs. Hoarseness of the cry is heard in laryngitis, and a hoarse, whimpering, and somewhat nasal cry occurs in congenital syphilis. A nasal cry is present also in coryza of other nature. A characteristic "brazen" cry is heard in spasmodic croup. A short cry of pain after coughing denotes pain, as in pneumonia or pleurisy. Crying is usually unattended by the production of tears until about the 3d month. After this date crying without tears indicates a condition of dangerous debility. A sudden shriek at intervals, without ordinary crying, uttered by a child in a stuporous state, suggests tuberculous meningitis ("hydrencephalic cry"). Sudden crying out at night may, however, be produced by the night-pains of disease of the bones. After infancy is passed violent, unappeasable crying, with which a child suddenly starts from sleep, is indicative of night terrors. In later infancy and childhood the cry of anger is often witnessed. It is loud, violent, without any piercing character, unattended by any evidence of pain, and generally associated with some evident reason for wrath. The infant while crying from this cause often stiffens itself all over, or throws its head backward; while the older child may stamp its feet, throw itself upon the floor, and even beat its head against the floor or wall. Finally, there is the very common and very deceptive cry occurring in infants who have in various ways not been well trained, which is merely an expression of discontent with their condition, although without anger or pain. A baby, for instance, awakens from sleep and cries violently. As soon, however, as it is taken up by the nurse its crying ceases, and smiles replace the tears.

Cough.—A short suppressed cough followed by a facial expression of pain is heard in pneumonia and pleurisy; a peculiar barking, brazen cough in spasmodic croup or the early stage of laryngeal diphtheria; a tight hoarse cough in laryngitis and tracheitis. Long, hard paroxysms of dry cough sometimes causing pain in the chest occur in the early stages of severe bronchitis, and a loose rattling cough in bronchitis after secretion is established. The long paroxysms of rapid, short expiratory efforts, continuing until suffocation seems impending and followed by a crowing inspiration, are characteristic of pertussis. In this disease mucus is often driven from the mouth. A very similar cough may occur in enlargement of the bronchial glands, yet not often accompanied by a whoop. It may be particularly troublesome at night, as the cough in pertussis is likewise. A peculiarly severe, ringing, brazen cough, in some respects resembling that of croup but often paroxysmal, is sometimes caused by the presence of glandular or other tumors or abscesses within the thorax, or the presence of a foreign body in the windpipe. An annoying "tickling," hacking cough occurs in pharyngitis, especially when the uvula is elongated. When the pharyngitis is severe the cough causes pain in the throat. A hard dry cough, often severe, is heard in passive congestion of the lungs produced by disease of the heart. Indigestion is frequently attended by a hacking cough, the so-called "stomach cough." Asthma has a short dry cough, not paroxysmal.

Voluntary expectoration of *sputum* following cough does not, as a rule, take place in any disease until 6 or 7 years of age. Sputum for examination must be obtained by introducing an elastic catheter or a pledget of cotton or cloth on an applicator to the base of the tongue. This occasions coughing, whereupon the sputum may be aspirated by the catheter or caught on the cloth.

Pain.—Pain, either subjective in origin or produced by handling, is of the most varied form and significance in early life. It has already been discussed to some extent under different headings. The determination of the seat of pain in infants and young children is often very difficult. Frequently tenderness can be ascertained by handling various parts, and observing whether a cry or a grimace is produced. Children under 5 years seldom locate pain exactly in attempting to describe it. Pain in the head is very common. It may indicate the onset of fever, whether this be of short or of more prolonged duration, being a frequent early symptom of typhoid fever and still oftener of meningitis. It is common in intracranial tumor. In other cases it is dependent upon coryza, anemia, dyspepsia, eyestrain, otitis, fatigue, excessive mental work at school, migraine, dental caries, nephritis or heart disease. Pain referred to the mouth may indicate stomatitis of various sorts, or toothache. In the throat it may attend many forms of inflammation there. Pain in the neck may be due to inflammation of the lymphatic glands, mumps, tonsillitis, or the tenderness of the muscles in torticollis. It may also be a symptom of caries or of basilar meningitis, especially when the pain is increased by motion. Pain in the thorax depends most frequently upon pleurisy and pneumonia. It may then be present only during cough. Occasionally pain in one side is produced by herpes zoster. Heart-disease is sometimes attended by severe attacks of precordial pain. Tenderness of the chest on grasping the child under the arms to lift it may occur in pleurisy, but is most marked in rickets, probably due to a scorbutic complication. The symptom may be deceptive, as the pain can be in reality located in some other part of the body, which the lifting has disturbed. Pain in the abdomen depends upon digestive disturbances, peritonitis, appendicitis, or intussusception, and is not infrequently a symptom of spinal caries. Inflammatory affections of the chest quite often produce a pain which is referred to the abdomen. Pain with stiffness on moving the back strongly suggests caries of the spine. In the limbs it may depend upon rheumatism or other form of arthritis, or on poliomyelitis, but in infancy is far oftener a symptom of scurvy. It becomes particularly evident when the child is handled. Pain is liable to attend inflammatory affections of the bones, among which may be mentioned periostitis and osteomyelitis. The existence of undiscovered fractures in infancy is a fruitful and puzzling source of severe pain. Hip-joint disease occasions pain which is referred to the thigh or, commonly, to the knee. Caries of the spine may occasion pain in the lower limbs. An unusual degree of local or general hyperesthesia is not uncommon in different nervous disorders.

Breath.—A rancid, butyric acid odor may be present on the breath of infants suffering from the vomiting of gastric indigestion. Acute febrile conditions or acute indigestion may produce the odor of acetone, or give rise to other odors of an unpleasant character. Ozena, ulcerative stomatitis, the accumulation of secretion in the tonsils, and the sputum from pulmonary abscess and from bronchiectatic cavities can produce a very offensive odor, while in gangrene of the lung and in noma the sick-

ening odor is almost unbearable. A stercoraceous breath is exceptionally noticed in intestinal obstruction.

Vomiting.—Vomiting is a symptom so frequent in early life and due to such varied causes that it will receive independent consideration later (p. 700). In this connection a few of its diagnostic indications may be mentioned. Simple regurgitation, without effort, of food which is little if any changed, occurs in the case of healthy infants who have taken more milk than they can comfortably hold, or who have been carelessly handled after feeding. True vomiting is accompanied by more effort and by evidences of nausea, such as pallor of the face and perspiration. It is very common at the beginning of acute febrile diseases in early life. When acute and of brief duration, accompanied by nausea and coated tongue, and perhaps followed by diarrhea, it is generally the sign of acute gastro-intestinal disturbance. Very obstinate vomiting, frequently with much mucus, occurs in chronic gastric indigestion and gastritis in infancy and in cases of stenosis of the pylorus. Vomiting may be the evidence of a toxic state, as seen, for instance, in uremia and in acute milk-poisoning. If repeated frequently for days, and attended by retracted abdomen, headache, moderate fever, and some degree of constipation, it is very suggestive of meningitis, or, in the absence of fever, of brain tumor. Recurrent vomiting is a disorder not to be forgotten. Vomiting is common after violent paroxysms of coughing, especially in pertussis and severe bronchitis. Very obstinate vomiting with distention of the abdomen and some degree of constipation occurs in peritonitis. When obstinate constipation is combined with obstinate vomiting, the existence of obstruction of the bowels, sometimes congenital, may be suspected.

Finally, the extreme ease with which vomiting is brought about in infants and in children must be borne in mind. This is especially true when once the tendency to vomit has been developed. Thus in many infants slight moving of the body after a meal is sufficient to cause the loss of it. The mere taste of the food may occasion vomiting, both in infants and older children, everything being ejected the taste of which is not liked. This is probably the reason why nourishment given to infants by gavage may be retained, when that entering the mouth in the usual way may be vomited.

Bowel-movements.—Alteration of the character of the feces is common in disease in infancy. A large amount of white, lumpy material in the stools, especially of bottle-fed infants, generally indicates too large an amount of fat in the food. Mucus appears very readily in the stools of infants. It may indicate merely a catarrhal process, but in large amount and accompanied by fever and straining suggests an inflammatory condition. It is generally present in considerable amount after the administration of a purgative, such as castor oil. Blood in the passages may denote a purely local process, such as fissure, hemorrhoids, congestion, or constipation. Mixed with mucus and attended by straining efforts it may be the evidence of enterocolitis or intussusception; and when in larger amount, without mucus, may be one of the symptoms of purpura hemorrhagica, severe ulceration, or rectal polypi. Black stools suggest the existence of hemorrhage in the upper intestinal tract. A pea-green color is often physiological, but the presence of dark-green slimy masses is very frequent in intestinal indigestion and in enterocolitis. Putty-colored stools may be due to deficiency in the secretion of bile, but oftener in infancy white or grey, formed or unformed stools are seen in those who

are taking much more butter-fat than they can digest. Hard, scybalous masses coated with mucus occur in older children with forms of intestinal indigestion of a chronic nature. Intestinal parasites of different sorts or their ova may frequently be discovered.

The *odor* of the stools during disease varies. In cholera infantum the passages are nearly odorless; in other forms of diarrhea in infancy they may be sour smelling and irritating to the adjacent skin, or may be very offensive, the difference in odor depending upon whether carbohydrate or protein indigestion is present. The administration of such substances as beef-juice and egg-water often produce exceedingly unpleasant odors. The action of certain drugs and food upon the color of the passages is to be remembered; iron producing a black, and bismuth a greenish-black hue, and such substances as hematoxylon and krameria, formerly much in use, a reddish color. Infants fed upon a high carbohydrate-diet, such as malt-soup or buttermilk-mixtures, exhibit smooth, brownish stools. A fuller discussion of the character of the feces in disease will be given later. (See p. 731.)

The *number* of passages varies greatly; and in estimating the importance of this matter, the character and amount of the food taken must be considered carefully. Constipation in infancy is sometimes a sign of a very thorough digestion which leaves little waste-material to pass; in other cases it may depend on the great length of the sigmoid flexure in early life; in others upon impaired general health, and in still others, when combined with a failure to gain in weight, it is caused by insufficient nourishment. On the other hand, an undue number of movements, unless diarrheal in character, may be merely a natural method of getting rid of an excess of nourishment. Frequent, diarrheal stools may be due to inflammatory conditions of the mucous membrane, or tuberculous or other ulceration; toxic influences, as in the infectious fevers; local irritation such as improper articles of food; or reflex causes, as seen in diarrhea after surface-chilling.

Urine. (See also Vol. II, p. 163.)—*Retention of urine* may be dependent upon the pain which urination causes, as in vulvitis, inflammation of the prepuce or glans, or the smarting produced by very acid urine. Sometimes it is the result of obstruction, as in cases of calculus in the urethra, very narrow foreskin, malformations of the urinary tract, or stone in the bladder. *Suppression* or very *great diminution* in the amount of urine may attend acute nephritis, acute fever, profuse diarrhea, severe vomiting, renal calculus, and sometimes intestinal obstruction. *Great increase* in the amount of urine occurs in diabetes, diabetes insipidus, after crisis in fever, after attacks of abdominal pain or of convulsions, and in some forms of chronic Bright's disease. *Albuminuria* is occasionally physiological. It occurs also in Bright's disease, in severe cardiac affections, in many febrile states, sometimes in scurvy, and from the admixture of pus or blood. *Blood* in the urine, unless in very small amount, makes it appear smoky, muddy or bright red in color. It may occur in Bright's disease, scurvy, in some grave cases of infectious fevers, purpura, stone in the bladder, and especially in stone in the kidney. Often the presence of blood manifests itself most noticeably by a stain on the diaper. Sometimes the altered color is due to hemoglobin and not to the presence of corpuscles. This is true of infectious hemoglobinuria of the new born. A paroxysmal hemoglobinuria is occasionally seen in older children. The urine is of a reddish-yellow color in jaundice. Uric acid sand may appear on the diaper of infants, while in other cases

an excess of urates makes the urine of infants milky in color. Pus in the urine is found in inflammation of any part of the urinary tract, as pyelitis, cystitis or urethritis. It also results from contamination by the discharge in vulvovaginitis.

Blood.—The significance of the alterations of the blood in disease depends upon the variation in the amount of hemoglobin, and in the number and character of the erythrocytes and particularly of the leucocytes the presence of pathological corpuscles and not infrequently the alterations shown by chemical examination. The matter can best be considered in connection with the various diseases of the blood (p. 000).

Serous Cavities.—By the puncture of the pleural cavity the existence of effusion is made known and the character of the fluid determined. The diagnosis between pneumonic consolidation and pleural effusion often hinges upon the result of this procedure. By lumbar puncture the existence of forms of meningitis can be positively determined in most cases. The diagnosis of the nature of an abdominal disorder may, in like manner, be aided by the employment of puncture in some instances.

The appearance and character of the fluid obtained by the puncture of serous cavities including the kind of formed elements present in it is shown by microscopical and chemical examination. The presence and nature of any bacteria found must receive careful consideration. The matter can better be studied in another connection. (See Pleurisy, Vol. II, pp. 102, 113, and Lumbar Puncture, Vol. II, p. 235.)

Mental and Nervous Symptoms.—In what has already been said incidental reference has been made to various diseased states of the nervous system. What follows is by way of addition or recapitulation. Further discussion will be found in the section upon Nervous Diseases (Vol. II, p. 232).

Various forms of mental defect are to be recognized. The mother often wrongly supposes that the child's failure to take notice, walk, or talk is due respectively to defective sight or hearing, paralysis of the legs, or tongue-tie. To determine whether failure to notice objects at the proper age is due to idiocy or to blindness is often a matter of difficulty. An ophthalmoscopic examination may settle the question. Slowness in learning to speak, when not dependent upon imbecility, can be the result of defective hearing or of a general slowness of development caused by ill-health. Yet often it seems to be merely a peculiarity of the individual, irrespective of other conditions, and need then cause no anxiety. A marked tendency to allow the saliva to dribble out of the mouth is a characteristic of the idiotic state. A condition of confusional insanity, almost maniacal at times, may develop after fevers, especially typhoid. Simple anger becomes so uncontrollable in many children that it occasionally seems almost maniacal. Delirium is a frequent attendant of high temperature in children, and does not necessarily indicate the presence of serious disease. It may occur even in infancy, although at this age convulsions tend to replace it. Convulsions, indeed, are very prone to develop in early life. They may be due to many causes, often of the most trivial nature. Pneumonia and all the infectious fevers, especially scarlatina, may be ushered in by them, and even high temperature from other sources is likely to produce them in some subjects. Rickets is a peculiarly frequent predisposing cause. They may result, too, from peripheral irritation of various sorts, particularly of the gastrointestinal tract, diseases of the brain, and uremia. Occurring in the newborn they are frequently the sign of intra-cranial trauma or disease.

Hysteria may be witnessed even in early childhood, and the presence of malingering dependent upon this is often to be recognized. In fact, the power of imagination is so great in childhood that pains are very liable to be complained of if the suggestion of pain is made to the child. Aphasia in children has not the grave import or the diagnostic value of the symptom in later life. It sometimes occurs temporarily after typhoid fever.

Psychic and nervous symptoms are much more easily evoked in early than in adult life. Great restlessness or excessive talking during sleep, somnambulism, and such semi-delirious states as are seen in night-terrors illustrate the readiness with which the mental condition of children is affected by such slight causes as temporary fever, indigestion and over-exertion. Extreme excitement, with crying, may be due to disease or may be dependent only upon accidental causes. Great irritability is evidence of an unhealthy state. Sometimes it may be merely the result of indigestion or malaise, but in other instances it points strongly to the beginning of meningitis of the tuberculous form. Indifference to surrounding objects and the ceasing to play with toys indicates a serious affection of health. It may depend upon great weakness or upon intracranial disorder. Stuporous and comatose states with the attendant symptoms occur in actual organic disease of the brain, or may be the result only of severe functional disturbance of it. Thus the condition of "pseudo-hydrocephalus," very closely resembling meningitis, may develop as an attendant upon great weakness, especially after severe diarrhea. Absence of the patellar reflex may occur in poliomyelitis, the muscular dystrophies, diphtheritic and other neuritic paralyses and in Friedreich's ataxia. Increase of the knee-jerk may be a symptom of cerebral palsy, Pott's disease, disseminated sclerosis and of some forms of cerebellar disease, or may be without serious import.

CHAPTER X

MORBIDITY AND MORTALITY

TENDENCY TO DISEASE IN INFANCY AND CHILDHOOD

The greatest tendency to disease in general appears to exist in the 1st year of life, and especially in the new born; the least at the age of from 10 to 15 years. There is besides, as has already been indicated (p. 182), a predisposition to certain diseases during infancy and childhood as contrasted with adult life, and a comparative freedom from others. A difference exists, too, in the susceptibility to particular diseases at the various periods of early life, different stages of development being attended by different maladies. Thus in the new born exists a group of affections, almost characteristic, which are due either, first, to disease or defects of development persisting from fetal life, or, second, to injuries or disease arising at birth or very shortly after it. In the first class are all the malformations and congenital disorders, such as those of the heart, intestines and brain, as also the different forms of imperfect development visible to the eye, and the inherited affections, especially syphilis. There is also the state of congenital asthenia with which very many infants appear to be born and which in no way depends necessarily upon premature birth, although a common result of this. In the second group are the injuries occurring at birth, which result from parturition, notably the forms of paralysis dependent often upon meningeal hemorrhage or direct injury of the brain or nerves, hemorrhage into some of the other visera, the various dislocations and fractures, and injuries of the surface of the body, such as cephalhematoma. In this class, too, are a number of diseases which are largely peculiar to the new born, such as forms of septic infection, atelectasis, pyloric stenosis, certain types of pneumonia, icterus, ophthalmia of the new born, etc.

From the end of the first 3 or 4 weeks of infancy, up to the end of the 1st year of life the prevailing diseases are those connected with the digestive apparatus, over one-half of them belonging to this category and being the result in most instances of artificial feeding. The nervous system is also unusually sensitive, and convulsive disorders are prone to occur. The general feebleness of the young infant and its deficient power of reaction render it less able to resist cold, excessive heat, and other debilitating influences. It is particularly in this year that children seem to fade away under the influence of torrid weather. Diseases of the respiratory tract are very common, and pneumonia is frequent and liable to be of the catarrhal form, and often fatal. Scurvy is seen especially at this time, as are the earlier manifestations of rickets. Affections of the thymus gland are to be noted. Acute infectious diseases, as a class, are less liable to occur, with the exception of pertussis and the septic infections, and to a less extent measles. Diseases of the skin are common, thrush is a frequent affection, and tuberculosis is often encountered, usually in the form of a general tuberculous infection. Inflammation of the lymphatic glands begins to be frequent, especially of the internal glands at this age, and may be either simple or tuberculous in nature.

The 2d year of life sees the respiratory diseases continuing to prevail, while the digestive disorders, though still frequent, are, on the whole, less so, and less fatal in their results. Convulsions still occur very readily from the slightest causes. Meningitis is common, rachitis extremely so, and pyelitis often seen. Diseases of the lymphatic glands, especially in connection with other disorders, continue to occur with a frequency characteristic of early life. Adenoid growths of the nasopharynx and hypertrophy of the tonsils become frequent. In this period, too, the marked disposition to the occurrence of acute infectious fevers, especially diphtheria, begins to be manifested. Throughout the first 2 years of life acquired affections of the heart are uncommon and rheumatism rare. Tuberculosis shows itself as tuberculous peritonitis, adenitis, meningitis, in the form of tuberculous bronchopneumonia, and very often as a widespread general tuberculous infection. Typhoid fever is relatively much less frequent than later, and its symptoms often obscure.

During early childhood; *i.e.* after the age of 2 years to that of 6 years, there is a predisposition to certain forms of digestive disturbances, particularly disorders of the stomach and small intestine. Diarrhea is less troublesome than in infancy. Tonsillitis, pharyngitis and various forms of stomatitis increase in frequency. Respiratory affections are very common, and spasmodic croup becomes one of the terrors of the household. Rheumatism and acquired affections of the heart begin to be observed although still not common. The tendency to the acute infectious diseases appears to be approaching its height. Appendicitis, rare under the age of 2 or 3 years, begins to be frequent.

In later childhood; *i.e.* after the age of 6 years, chorea becomes frequent and rheumatism and diseases of the heart also. Functional neuroses dependent upon school-life are to be noted, as are also myopia, scoliosis, headache, anemia, and the like. The infectious diseases continue extremely frequent. Meningitis is of common occurrence, and various psychoses appear as puberty is approached.

Chronic affections of the kidney, with the exception of pyelitis, are infrequent during the whole of infancy and childhood, unless as a result of scarlet fever. Acute nephritis may readily attend the infectious diseases, and often in infancy is associated with severe diarrheal disturbances. Disease of the bones and joints, generally of a tuberculous nature, is frequent in all periods of early life, although less so in infancy than in childhood. Disease of the brain-substance is rare except when secondary to meningeal disturbance, and that of the spinal cord, nerves, and muscles likewise so, the exceptions being poliomyelitis, which is common in infancy and early childhood, the neuritis following diphtheria, the transverse myelitis resulting from spinal caries, and the muscular dystrophies and certain systemic nervous affections which are characteristic of childhood.

FETAL MORTALITY. STILL AND PREMATURE BIRTHS

Fetal mortality, including miscarriages and still-births, shows a high percentage of the total number of conceptions. Whitehead¹ calculates the proportion of miscarriages to pregnancies as 1 : 7, and Priestly² as 1 : 4½. In 28 cities of the United States in 1890³ the number of still-births bore

¹ Ref. Ballantyne, *Disease of the Fetus*, 1892, I, 8.

² Ballantyne, *loc. cit.*

³ U. S. Census, *Vital Statistics*, II, 142.

to the number of children born alive a ratio of 62.65:1000; i.e. 5.9% of the total births were still-births. An estimate of the European states makes the still-births 3.79% of the total births (Wappäus).¹ Ruppin² gives the still-births of Prussia during 25 years as 3.69% of the total births.

Premature birth is also a frequent cause of death during infancy. This might, in a sense, be included in fetal mortality, since death finally occurs because the organism is not in a state fully prepared for life outside the uterus. The Census statistics of the United States for 1890³ make 5.68% of all the deaths under 1 year dependent upon premature birth. This is probably below the actual figures, since many deaths attributed to inanition and similar causes might properly have been classified with those resulting from premature birth.

MORTALITY IN INFANCY AND CHILDHOOD

Death-rate Especially in the First Year.—It is a matter of common observation that the younger the child the more dangerous, as a rule, is any disease affecting it. The exhaustive studies of Eröss⁴ based

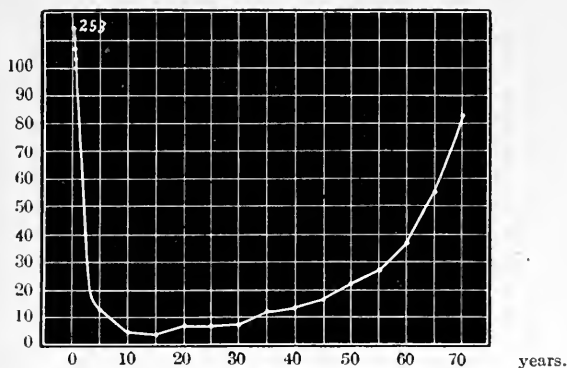


FIG. 29.—GRAPHIC CHART REPRESENTING THE NUMBER OF DEATHS FOR DIFFERENT YEARS FOR EVERY 1000 PERSONS OF EACH AGE.

The figure 253 represents the number of still-births. (Westergaard, *Die Lehre v. d. Mortalität.*, 1901. Ref. Prausnitz, in Pfandler and Schlossmann's *Handb. der Kinderheilk.*, 1906, I, 1, 279.)

on the vital statistics of 13 European States for a number of years, showed that an average of 18.33% of all children born died in the 1st year, equaling 26.89% of the total mortality for all ages. Of the deaths in the 1st year, more occurred on the 1st day than on any other. Of the fatal cases in children under 1 year, 31.86% took place in the 1st month, and 73.13% in the first 6 months. Nearly one-half of the fatal cases of the 1st month were in the 1st week. Later investigations support these figures, those by Holt and Babbitt,⁵ showing that of 100 deaths in the 1st year, 13 occurred on the 1st day, 22 in the 1st week, 28 in the first 2 weeks, and 33 in the 1st month. All this shows a rapidly decreasing mortality in proportion as the infant grows older. This diminishing mortality, with

¹ Allg. Bevölkerungsstatistik, 1859. Ref. Pfeiffer, Gerhardt's *Handb. d. Kinderkr.*, I, 551.

² Schmidt's *Jahrbücher*, 1902, CCLXXIII, 233.

³ *Loc. cit.*, IV, 666.

⁴ *Zeitsch. f. Hygiene*, XIX, 371.

⁵ *Journ. Amer. Med. Assoc.*, 1915, LXIV, 287.

subsequent increase as adult life advances, is illustrated graphically in the accompanying curve (Fig. 29) after Westergaard.¹

The statistics of the United States for the census year ending in 1890² give very similar results regarding the high mortality of the 1st year. A percentage of 18.29 deaths occurred in the 1st year of every 100 born alive in the Registration area.³ During 6 years' observation in this area 251,424 deaths occurred under 1 year, a percentage of 29.71 of the total number of deaths for all ages. Of these deaths in the 1st year 55.77% were in the first 3 months, 18.19% at from 3 to 6 months, 14.35% from 6 to 9 months and 11.62% from 9 to 12 months.

There is considerable variation in the mortality rates according to *geographical distribution*, the relative number dying in the 1st year in the countries studied by Eröss being lowest in Ireland (9.4%) and Sweden (9.7%) and 3 times greater in Saxony (28.1%) and Bavaria (28.7%). It is still higher in Russia (32.6%) (Gundobin).⁴ Johannessen's⁵ statistics for Norway gave a mortality of 9.8%.

The influence of *crowding and bad hygiene* is very positive. The infant-mortality is higher in the poorer classes and is constantly greater in cities than in the country. Many of the larger cities of Europe and America have shown a mortality in the 1st year of from 20 to 30 per cent. of those born (Deutsch).⁶ The mortality is nearly always greater among illegitimate children as a class, for various reasons not dependent on the innate vitality of the child. Deaths among infants in Homes, Asylums, and Hospitals are extremely numerous, in spite of all efforts to prevent this. In some of the poorly managed institutions the mortality has reached, or even exceeded, 90 per cent., and in the best it has often averaged 50 per cent. or 60 per cent. The death-rate is, however, being decidedly reduced as improved methods of hygiene and diet are being followed. The causes of the high mortality depend partly on the character of the infants in these institutions, partly on the improper care given in some of them, partly on the spread of disease from one child to another, and largely on a general, imperfectly understood, deleterious influence which attends the keeping of many infants together in one place, and to which the title "hospitalism" is often applied.

The influence of *artificial feeding* on mortality in the 1st year is very striking. Of 34,325 infants under one year dying in Berlin, 17.7 per cent. were wholly or partially breast-fed, and 82.3 per cent. entirely artificially fed (Eröss). Undoubtedly the feeding was in most instances improperly managed. The statistics for Munich, as published by Frank⁷ gave very similar results: 85.22 per cent. of 8329 deaths under 1 year of age being in artificially fed infants, with but 14.78 per cent. among those breast-fed.

The influence of *season* upon the mortality is decided. Both the coldest and hottest seasons of the year are attended by high mortality,

¹ Die Lehre von der Morbidität und Mortalität, 1901. Ref. Prausnitz, Pfaundler und Schlossmann's Handb. der. Kinderheilk., 1906, I, 1, 279.

² Loc. cit., I, 22.

³ The "Registration area" for that date included the registered states, Massachusetts, Connecticut, New Hampshire, Vermont, Rhode Island, New York, New Jersey and Delaware; the District of Columbia, and 83 registration cities in non-registration states.

⁴ Jahrb. f. Kinderh., 1898, XLVIII, 368.

⁵ Jahrb. f. Kinderh., 1902, LVI, 259.

⁶ Centrallbl. f. Kinderh., 1903, VIII, 269.

⁷ Ref. Jacobi in Gerhardt's Handb. der Kinderkrankh., I, 319.

especially in the 1st year of life. In the United States the mortality under the age of 5 years is very much the greatest in the hot summer months.¹ The increase of infantile mortality during the hot weather is not only dependent upon the deleterious changes which more readily take place in the milk at this season, but to a considerable extent upon the direct influence of the torrid weather upon the infant. This has been carefully demonstrated by Rietschel,² Schereschewsky³ and others. The plotted curve shown in the figure (Fig. 30), after Rietschel, is of the infantile mortality in Munich from digestive diseases compared with the air-temperature in centigrade. A partial exception to the fatal effects of hot summer weather exists in the 1st month of life, greatly debilitated children being often favored by hot weather.

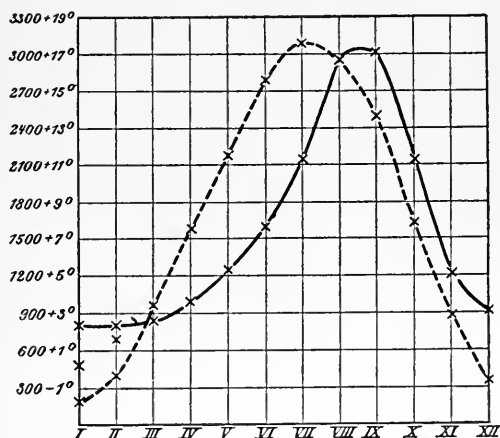


FIG. 30.—DEATHS FROM DIGESTIVE DISORDERS IN INFANTS IN MUNICH FROM 1895 TO 1904.

The interrupted line indicates the mean air-temperature; the other the number of fatal cases for each month. It is to be noted that the height of the death-curve occurs about 4 weeks later than that of the temperature-curve. (Rietschel, *Ergebn. d. inn. Med. u. Kinderh.*, 1910, VI, 416. Based upon statistics of Fürst, *Vierteljahrschr. f. öffentl. Gesundheitsflege*, 1907, XXXIX, 417.)

Sex exerts some influence. In 12 states of Europe 55.59 per cent. of the deaths in the 1st year were among males and 44.41 per cent. among females (Eröss). In the 6-years' record of the Registration Area of the United States⁴ 54.83 per cent. of the deaths under 1 year were among males and 45.17 per cent. among females. As, however, there are about 95 females borne for every 100 males, the death-rate of females as compared with the males born is a trifle greater than the figures express.

It is difficult to determine the influence of *race* apart from attendant climatic, hygienic, and similar conditions. In the United States the mortality of the white races in the 1st year is greatest in the Italians, Bohemians, and Hungarians. Among the dark races of the United States (Negroes, Chinese, and Indians), the mortality in the 1st year exceeds greatly that of the white infants.

¹ XI Rep., U. S. Census, 1890, II, 63.

² *Ergebnisse der inn. Med. u. Kinderh.*, 1901, VI, 369.

³ *Arch. of Ped.*, 1913, XXX, 916.

⁴ *Loc. cit.*, IV, 666.

Diminution in the Death-rate.—A very interesting question, especially as regards infants, is whether or not the death-rate has diminished in recent years under increased knowledge of infantile diseases, and especially of those of the digestive apparatus. There appears to be good reason to believe that a lessened mortality-rate has indeed occurred. Rich¹ in a comparison of the statistics of the United States Census of 1900 with that of 10 years earlier, pointed out that the infantile death-rate in the Registration-Area had decreased from 20.58 per cent. per 100 births in 1890 to 16.54 per cent. in 1900; and that while the death-rate of children from 1 to 5 years of age in New York City in 1891 equalled 96 per 1000 individuals of this age, in 1896 it had fallen to 77.5 and in 1900 to 67 per 1000. Further, according to the statistics of Baker² the death-rate in infants in New York City, which was 289 per 1000 infants living in 1880, gradually but uninterruptedly fell to 134 in 1910. This history of a falling death-rate in infancy and early childhood is borne out by the experience of many other cities of the United States, the varying degree depending largely upon the amount of civic and professional care observed. The Statistical Report of the New York Milk Committee upon the infantile mortality for 150 cities of the United States for the year 1916 showed a mortality for the 1st year which averaged in the neighborhood of 10 per cent. or slightly less. The reduction in infantile mortality appears to be taking place especially in subjects suffering from gastrointestinal diseases. This has been due to a large extent to the improvement in the quality of the milk supplied and the more strict supervision maintained in cities during the summer season, and to a better knowledge of the proper treatment of digestive disturbances.

Causes of Death at Different Ages.—Among the most frequent causes of death in the 1st year various conditions appearing to indicate an inborn state of debility occupy one of the most prominent places. Of the 174,614 deaths in this period occurring in Ireland during 10 years "Debility, Atrophy, Inanition" headed the list with 29,136 deaths, or 16.69 per cent. "Convulsions" came next with a percentage of 11.89 (Langford Symes).³ The latter, however, and to some extent the former, represent terminal diseases or methods of dying rather than the real cause of death. The mortality-statistics of Massachusetts during 5 years (1892-1896) made diarrheal diseases responsible for 29.49 per cent. of the total deaths occurring in the 1st year of life (S. W. Abbott),⁴ and respiratory diseases, classified as "pneumonia" and "bronchitis," for 11.97 per cent. The deaths attributed to "atrophy" and "debility," many of which were probably the results of gastrointestinal disease, equalled 10 per cent. The statistics of the Registration Area of the United States for 1913, as analyzed by Woodward⁵ made diseases of the digestive system responsible for deaths in the 1st year of life in 27.12 per cent. and diseases of the respiratory system in 15.85 per cent. Of the latter 96 per cent. depended upon bronchitis or pneumonia.

The mortality from digestive diseases is especially large in cities. 38.5 per cent. of the deaths under 1 year occurring in the civic population of France were due to gastroenteritis (Budin).⁶

¹ Arch. of Ped., 1905, XXII, 762.

² 15th Intern. Cong. of Hygiene, 1912, III, 3, pt. 1, 139.

³ Med. Mag., 1898, June.

⁴ Jour. Mass. Assoc., Board of Health, 1898, Dec.

⁵ Amer. Jour. Obstet., 1916, LXXIII, 362.

⁶ Ann. méd. et chir. inf., 1903, VII, 181.

Of the chronic infectious diseases syphilis is more fatal in the 1st year than at any other time, about $\frac{1}{2}$ of all the deaths from this affection occurring in this year. Tuberculosis is as common a cause in the 1st year as later.

The number of deaths from the acute infectious fevers is generally less than later in infancy and childhood owing to the much smaller incidence. To this, however, pertussis and erysipelas form exceptions, both of them causing more deaths in the 1st than in any other year. The mortality from measles is also high at that period. The cases of the other infectious fevers, although much fewer, have a relatively unfavorable prognosis.

In the 2d year the mortality is much less. The Massachusetts' statistics (S. W. Abbott)¹ for 5 years gave about $\frac{1}{5}$ as many deaths for the 2d year as for the 1st year of life. Gastrointestinal diseases and respiratory affections are the most prominent causes, the latter gradually growing in importance and the former lessening. The deaths from gastrointestinal affections equalled 19.79 per cent. of the total deaths between 1 and 2 years of age, and respiratory diseases (pneumonia and bronchitis) 21.27 per cent.

Infectious fevers, particularly scarlet fever and diphtheria, play a more important part in this year, rubeola being about as frequent a cause of death as before. The deaths from tuberculosis in various forms are numerous, especially from general tuberculosis and tuberculous meningitis. Convulsions still remain a very prominent cause, or, rather, method, of death.

In *early childhood*, from the age of 2 to that of 5 or 6 years, deaths are chiefly due to the infectious fevers. According to the XI Census Report of the United States² the number of cases dying of scarlet fever from the age of 1 to 5 years, as compared with the total deaths at all ages from this disease, equalled 66.87 per cent. The corresponding percentage for measles was 64.36 per cent., for diphtheria and croup 64.09 per cent. and for pertussis 43.31 per cent. By far the largest actual number of deaths from these diseases was due, however, to diphtheria and "croup," the latter being without doubt generally diphtheritic.

Viewing infancy and early childhood together it is noticed that the mortality for different diseases reaches its maximum at different *seasons* of the year. A general average irrespective of the nature of the diseases shows the proportion of deaths in children to be greatest in the cool season of the year (Uffelmann).³ In some regions, however, the mortality is much higher in summer. This is very decided, for instance, in the United States⁴ for children under 5 years of age. The nature of the disease, however, has an important influence. Thus in the Metropolitan District of the United States during the 6-year record there were 16,413 deaths from diarrheal diseases in July against only 553 in December, while from respiratory diseases there were but 1921 deaths in August against 5102 in January. Sex continues to exert a slight influence after the 1st year to the end of early childhood, rather more males than females dying.

In *later childhood*; i.e. from the age of 5 or 6 years to that of 10 or 12 years the mortality is lower than at any other period of life. The

¹ *Loc. cit.*

² Pt. IV., 666.

³ Handb. d. Hyg. des Kindes, 1881, 85.

⁴ U. S. Census, 1890, II, 63 and I, 195.

principal causes of death at this time are the acute infectious diseases, especially diphtheria and scarlet fever. Respiratory diseases also constitute a very common cause; diarrheal diseases much less often so. Tuberculosis is still a frequent factor, showing itself often as tuberculous disease of the bones, lungs, and peritoneum; as well as of the meninges and in the form of general tuberculosis as before. Diseases of the heart now become a more prominent cause than before. The relative importance of certain of the diseases affecting early life, in their influence upon the death-rate, is well shown in the accompanying table given by Veeder¹ (Fig. 31) based upon 930,067 deaths in subjects under 10 years of age.

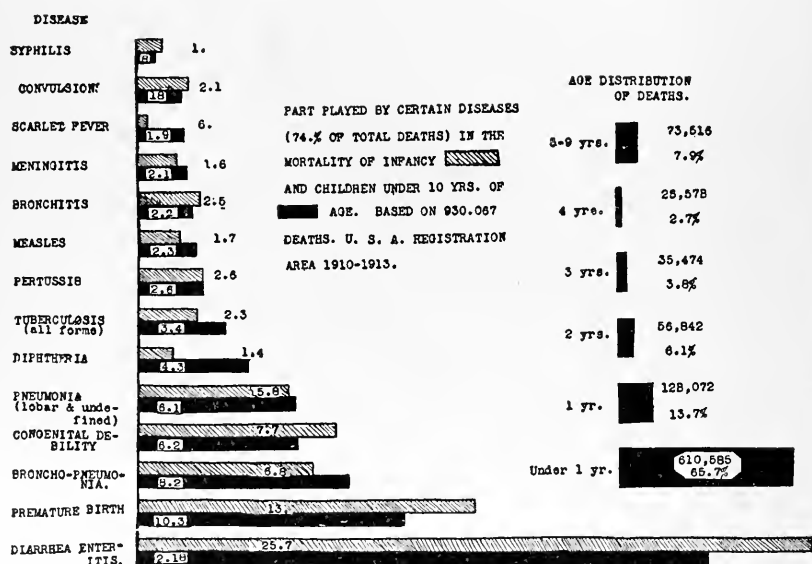


FIG. 31.—RELATIONSHIP OF CERTAIN DISEASES TO THE MORTALITY OF INFANTS AND CHILDREN.

(Veeder, *Arch. of Pediat.*, 1917, XXXIV, 322.)

Sudden Death.—Death occurring suddenly and unexpectedly is not infrequent, especially in the first 2 years of life, but the cause is often very obscure. In some instances the child has appeared in perfect health; in others the death occurred during some debilitating disease, yet without there developing any reason for the sudden fatal ending. Sometimes there had been some easily overlooked symptoms, which would probably have made the diagnosis possible and accounted for the death had a physician been in attendance. In some cases an autopsy reveals the cause, but in many it does not. The feeble resisting power of infancy and the great excitability of the nervous system are the principal reasons for predisposition to sudden death at this age. The various causes have been reviewed by Thiemich,² Vipond,³ and others.⁴

Conditions affecting the respiration are generally considered as holding the most prominent etiological place. Coryza in the new born may

¹ *Arch. of Pediat.*, 1917, XXIV, 322.

² *Vierteljahrsh. f. gerichtl. Med.*, 1901, XXI, 300.

³ *Montreal Med. Journ.*, 1901, XXX, 23.

⁴ See also article by the Author, *Amer. Med.*, 1903, V, 989.

rarely be the cause through what has been described as "aspiration of the tongue" (Bouchut).¹ By the violent efforts at breathing through the mouth the tongue is drawn backward and its under surface and tip become pressed against the hard palate, cutting off more or less the entrance of air. Death in the same way is stated occasionally to occur in young infants with pertussis. Asphyxia from over-lying probably does not happen as frequently as once supposed. Although a considerable number of infants die in this way, most of the cases assigned to this category probably belong elsewhere. Enlargement of the uvula has been designated a cause of fatal closure of the glottis, but the occurrence of the accident seems doubtful. Spasm of the glottis is perhaps one of the most frequent apparent causes; certainly so in those infants who had seemed to be in perfect health. This laryngo-spasm depends on the very great irritability of the nervous system which, although oftenest seen in debilitated, and especially rachitic children, exists sometimes quite independently of these conditions and is a symptom of spasmophilia (Vol. II, pp. 249, 256) and probably closely associated with lymphatism (p. 632). It is likely that many cases of death attributed to other causes in reality depend upon this state. Thus it seems likely that the sudden death assigned to enlarged thymus gland is not in fact connected with this, but with the lymphatism of which the thymus enlargement is but a symptom. (See Diseases of the Thymus Gland, Vol. II, p. 520; Lymphatic Diathesis, Vol. I, p. 632.) Whether or not the death is actually due to laryngo-spasm or depends upon sudden cardiac failure is still an open question. Perhaps each factor may be the cause in different cases.

Central respiratory involvement, or what seems probably such, sometimes occurs in cases presumably with the neurotic tendency which might in other instances lead to laryngeal spasm. In these cases the child may die as though from sudden heart failure. In other instances respiration becomes more and more rapid without other symptoms or discoverable reason, and the infant dies in a few hours. I have seen this accident occasionally. The cause of the condition is entirely obscure. A rapidly developing bronchopneumonia, especially in the new born, may sometimes kill with apparent suddenness, the attendants having noticed no symptoms.

Sudden death in atrophic infants is of comparatively frequent occurrence. Thus it often happens that an infant who has been ill for days or weeks with unchanged evidences of debility will unexpectedly be found dead in bed without there having been any alteration in symptoms. This is common also in cases of premature birth, autopsy often showing no lesions whatever. Yet in many of these cases, the cause is found to have been atelectasis, to which very young infants in a debilitated state are greatly disposed. In many other instances of atelectasis, however, the process comes on much more gradually and with evident symptoms. In some cases the deaths in marantic states may be due to the depressing effect of chilling or to a fall of body-temperature without discoverable cause.

Death with hyperpyrexia is often very unexpected in children who have been perfectly well or only slightly ailing but a few hours before. This may be seen in heat-stroke in infants, in malignant eruptive fevers, acute sepsis, and, still more frequently, in pneumonia in early life. Rarely sudden fatal asphyxia may result from the rupture of a caseous bronchial gland or of a retropharyngeal abscess into the respiratory tract.

¹ *Mal. des nouveau-nés*, 1885, 279.

In other cases it is due to pressure of intrathoracic growths, abscesses, or enlarged glands upon the pneumogastric nerve. The aspiration of food into the windpipe after vomiting has been assigned as a cause. It probably occurs much less often than has been supposed, and only in infants so weak that the ability to cough has almost disappeared. Instances of asphyxia from the entrance of ascarides into the larynx have been reported, but in most of these it is probable that the worms entered the respiratory tract after death. Edema of the glottis is also an occasional cause.

Sudden death from heart-failure is not infrequently seen in convalescence after diphtheria. It may also occur in debilitated states or in respiratory diseases, especially pleural effusion, as a result of too sudden a movement, improper position, or excitement. The same sudden cessation of the heart's action may take place in acute nephritis on account of the strain of increased arterial tension. Sudden stopping of the heart may occur in chronic valvular disease or as a result of distant nervous influences. It is probable that the sudden death which has occasionally followed quick movement or excitement in apparently healthy children, such, for instance, as the tossing of the child into the air, or which has occurred after exploratory puncture of the pleura, the giving of a hypodermic injection, or the administration of an anesthetic, has been in reality caused by inhibition of the heart's action in subjects with lymphatism. Many of the deaths assigned to spasm of the larynx are very possibly the result of this sudden stopping of the heart.

Gastrointestinal affections seldom occasion sudden death except indirectly. A number of cases are reported where it appears to have been induced by the irritation produced by large numbers of ascarides in the intestine. This is probably, however, of very rare occurrence.

Convulsions are a very common cause of sudden death in children already ill or in those in whom no disease has been detected. Nervous reflex irritation from various regions of the body may be responsible for them. Congenital syphilis is a not infrequent cause of sudden death, even in infants who appear perfectly healthy (Fournier).¹ Various malformations and accidents not already mentioned may also produce it, among them perforation of the intestine, entrance of foreign bodies into the larynx or trachea, injuries from forceps, strangulated hernia, external hemorrhages from the stomach or bowels, and internal hemorrhages, especially in the new born, into the suprarenal bodies and occasionally into the brain or elsewhere.

¹ La sem. m  d., 1901, XXI, 20.

CHAPTER XI

THE THERAPEUTICS OF INFANCY AND CHILDHOOD

Therapeutics in infancy and childhood possesses a few characteristics which sometimes render the subject easier and more satisfactory than in later years. All the tendencies of life are toward recovery, and the system generally responds well to remedial measures. Drugs when given should be in sufficient strength to do good, but never to do harm, and they should not be administered at all unless distinctly indicated. Under-dosing is futile; over-dosing harmful. To the latter there exists a widespread tendency among physicians. In the large majority of cases little medicine of any sort is required, and the careful attention to hygiene and diet is sufficient, with possibly such slight aiding of nature as the giving of a laxative, a diuretic, a warm bath, rest in bed, and the like.

Despite what has just been said the therapeutics of childhood for the most part presents many difficulties to the inexperienced. These depend chiefly on the fact that the child does not react toward remedial measures merely as a small-sized man would do, but has in many respects, its own susceptibilities. Some methods of treatment influence the system more powerfully than in adult life; others, useful in adults, are not so in childhood. The difficulty in giving medicines to many children is another factor. Numerous children, as a result of whim, or of the unpleasant taste or smell of a medicine, refuse this altogether. The administration of it may do harm through the struggling which arises, or may be always followed by vomiting. The taste of the same medicine may be not at all disagreeable to another child.

The therapeutics of early life is thus conveniently divided into A, *Administration of medicines by the mouth*. B, *Treatment other than by drugs by the mouth*. The order of consideration by no means indicates the relation of their importance, but rather the reverse of this. It may be said in passing that the older method of measuring by apothecaries weights and measures will be chiefly referred to, not as preferable, but because it is at the present time much the most familiar, and because there seems to be no practical advantage in writing prescriptions in the metric system and ordering the administration of the medicine in drops of fluidrams. Metric equivalents are given in the parenthesis whenever this seems best. A fluidram is equivalent to practically 4 c.c., a fluidounce to 30 c.c., a grain to 65.0 milligrams, and a dram (apothecary) by weight to nearly 4 grams. We are usually obliged in family practice to regard a teaspoonful as equalling 1 fluidram and a tablespoonful as the equivalent of $\frac{1}{2}$ a fluidounce; although the great variation in the size of teaspoons and tablespoons renders this inaccurate. A small glass graduate should always be recommended. Locke¹ gives a teaspoonful as equalling 5 c.c. and the tablespoonful as 15 c.c.

¹ Food Values, 1916, 24.

A. ADMINISTRATION OF MEDICINES BY THE MOUTH

1. Method of Giving Medicine.—In this, as in all forms of medical treatment in early life, we must as far as possible avoid the occasioning of fright, pain, or great excitement of any sort. Previous good training by the parents will have rendered the administration of medicines much easier. The physician, on his part, must see first, that the doses are small; second, that the taste is as pleasant as it can be made; and third, and most important, that the giving of any drug is really necessary. There is no question that children are often much over-dosed. It is often impossible to conceal a disagreeable taste. In such an event force may become necessary in giving the dose. Whether this shall be employed, or whether the treatment by drugs shall be foregone, depends upon the needs of the case. Judgment must be exercised, remembering,



FIG. 32.—MEDICINE-DROPPER.

Showing the correct method of dropping from the thicker portion of the tube.

as stated, that the harm from the fear and excitement may much exceed the good which the medicine might do. This scarcely applies to the cases of less severe ailments in vigorous children, when the refusal depends merely on ill-temper or obstinacy.

Where a dose of medicine has to be administered by force, the child should be wrapped as for the examination of the throat (p. 187) and held by an attendant, and the nostrils compressed for a moment. Generally the mouth opens and the spoon may be inserted to the back of the tongue, emptied, and slowly withdrawn. There is difficulty in swallowing if the spoon is not removed from the mouth. In infants, the pressing of the chin downward and backward may be sufficient to open the mouth. Often an infant who spits out the greater part of a teaspoonful of medicine will take it very well if it is given in divided portions. It may well be administered from a larger spoon, as there is less danger of spilling it should the child struggle.

The attendants should be told that, in cases where medicine must be given it is better to waste little time in pleading and in argument, but to

use firmness and decision from the very beginning. The child must in no case be deceived regarding the character of the medicine, as its confidence is lost thereby, and the giving of the next dose will be only the harder. In stuporous, delirious, or greatly exhausted conditions liquid medicine may be administered with a medicine dropper while the child is on its back. This should be inserted between the cheek and the teeth to prevent its being bitten. Nauseating remedies should not be given upon an empty stomach when this can be avoided.

In the ordering of quite small doses, or in the prescribing of powerful drugs, the physician must carefully remember the difference between the minim and the drop of many solutions, particularly of alcoholic ones, such as tinctures; and that the size of the drop also varies with the mouth of the bottle or with the dropper from which it is obtained. Consequently it is best to reckon the dose in minims rather than drops. Where the attendants are to measure the mixture a minim glass should be used, or, if drops are ordered, the nurse should be told exactly the method of dropping to be employed. The small, sharply-pointed medicine-droppers furnish a drop scarcely more than half the size of one obtained from the edge of the bottle-mouth. A curved dropper should be used, and the drop should fall from the thicker portion (Fig. 32). It is important, too, to recognize the differences in the sizes of spoons, which make it safer to have the "teaspoonful" or "tablespoonful" prescribed measured as a dram or a half ounce in an accurately graduated medicine-glass. (See p. 219.)

Unless really necessary those medicaments should not be used which cause unpleasant secondary effects, sometimes worse than the disease itself. Those, too, are to be avoided, when possible, which are almost uniformly disliked by children; and the individual tastes of the patient should be studied as well. Much can be done to disguise unpleasant tastes to a certain extent. As a rule, infants and children like sweetened mixtures, and syrups, saccharin or glycerine may be used for sweetening. Among the serviceable syrups for this purpose especially for older children are syrup of ginger, syrup of raspberry (*rubus idæus*), syrup of chocolate, syrup of orange-flower, syrup of wild cherry, syrup of vanilla, and syrup of lemon. For infants the simple syrup of the *Pharmacopeia* is to be preferred. The aromatic waters are well-liked, such as peppermint water, spearmint water, cinnamon water, and orange-flower water. For infants spearmint water (*aqua menthæ viridis*) is to be preferred to peppermint water (*aqua menthæ piperitæ*) since the sharpness of the latter is often unpleasant and occasions choking unless well diluted. The elixir aromaticus and the elixir glycyrrhizæ (elixir adjuvans) are very serviceable for disguising an unpleasant taste in the case of older children. It must not be forgotten, however, that these contain about 25 per cent., of alcohol and are not suitable for employment in full strength in the case of young subjects. Bitter medicines are generally disliked by children, but infants often appear to find them not unpleasant, especially if an abundance of sweetening is added. In this point there is the greatest individual difference seen. In some instances the bitter taste appears to make the infant vomit promptly. Nearly all liquid medicines may be sweetened and diluted with water when given, if the child finds the dose as prescribed disagreeable. Instruction on this point should be included in the directions upon the label on the bottle. An exception exists where sugar may disagree with the digestion; in the case of oils or emulsions; or where the addition of water makes the larger bulk still more difficult of administration.

The taking of a sip of water, milk, or orange juice, or the sucking a mint drop or chocolate immediately before medicine is taken dulls the sense of taste to some extent. Another sip immediately after washes away the unpleasant taste. The mother should be instructed that the employment of sweets in this way is to be confined to the occasions of giving medicine. Quinine may be disguised to a certain extent by licorice, or still better by syrup of Yerba santa or syrup of chocolate. A nearly tasteless preparation such as aristochin may replace it to a considerable extent. Castor oil should be given in emulsion with an aromatic water; floated on ice water, lemon juice, sarsaparilla or whiskey and water, or stirred in hot milk. By the latter method it is rendered nearly tasteless. The child must, of course, not be told that it is milk. In the case of infants it may be administered warmed in the spoon to make it less thick. Many infants always vomit it, and to these it should not be given at all. Others do not appear to dislike it in the least. To older children it is generally distasteful unless disguised. Cod liver oil, if disguised, is generally liked by children. It is best given as an emulsion flavored with mint, or combined with a syrupy malt-extract.

Drugs in powdered form, if comparatively tasteless and small in amount, may be placed directly upon the tongue, and washed down with a sip of water or milk. It is still better to have them combined with sugar when prepared. Powders of larger size may be mixed with a teaspoonful of jam, preserves, scraped apple, or the like, if there is no contra-indication. Entirely tasteless powders may conveniently be given on milk-toast or bread-and-milk and not be perceived at all.

Little children cannot take pills. Those of 3 or 4 years of age, if well trained, will swallow them readily if they are small. If the substance is bitter it should be sugar coated, or the drug enclosed in capsules. The pill may be conveniently offered in a small portion of jam or of preserved or fresh fruit. The rule already expressed still holds good; that no deception is allowable, and that the child should always be told that it is "medicine."

The passage of drugs to the infant through the mother's milk has already been alluded to (p. 106). For practical purposes in infantile medication this need not be considered, although the method is sometimes useful for the treatment of the fetus.

2. Dosage.—Various posometric tables have been constructed for use in childhood, and various methods proposed for the calculation of the proportionate doses as compared with those of adults. No fixed rule, however, can be entirely accurate. This is both because the relative susceptibility to different drugs varies greatly at this time of life, and because one cannot be guided either by age or by size alone. The rule of Clarke makes the weight of the child the numerator and 150 pounds the denominator, the resulting fraction being the portion of the adult dose to be employed. However, a child of 6 months weighing no more than a child of 1 month may need a decidedly larger dose than its weight would indicate. The rule of Young, which adds 12 to the age and divides the age by the result, ignores entirely the elements of size and weight. In practice, both age and weight must be considered. For the average child, Young's rule is a very convenient one down to the age of 2 years. Earlier than this the dosage may be based partly upon the age and partly upon the weight. The following table of proportionate dosage is the one I am in the habit of employing as a guide, but in a general way only.

TABLE 66.—PROPORTIONAL DOSES

Adult.....	1
18 years.....	$\frac{3}{4}$ +
12 years.....	$\frac{1}{2}$
8-10 years.....	$\frac{2}{3}$
6 years.....	$\frac{1}{3}$
4 years.....	$\frac{1}{4}$
3 years.....	$\frac{1}{5}$
2 years.....	$\frac{1}{7}$
1 year.....	$\frac{1}{10}$
9 months.....	$\frac{1}{15}$ or $\frac{2}{3}$ dose for 1 year
6 months.....	$\frac{1}{20}$ or $\frac{1}{2}$ dose for 1 year
Birth to 3 months.....	$\frac{1}{30}$ or $\frac{1}{3}$ dose for 1 year

3. Effect of and Susceptibility to Certain Drugs.—Certain medicines are peculiarly well tolerated and of much value in early life. Others are dangerous except in amounts smaller than the proportionate dose for the age would be. The dosage in general will be found in the table on p. 229. The following drugs require special mention.

Alcohol in the form of wines and spirits is tolerated in relatively large doses, and is exceedingly useful in many conditions. It is given far too frequently and freely by many physicians, and in absurdly small amounts by others. Judgment is, of course, required in deciding upon its employment and upon the dosage. It is indicated in many conditions where cardiac failure threatens, as in bronchopneumonia and in many cases of the infectious eruptive fevers, but especially in diphtheria. In exhaustion from any cause it is useful, as also in many cases of slight debility where a gently stimulating effect is sought. It should not be given in acute febrile conditions of short duration where the heart's action is strong and the temperature high. It is not needed in mild cases of typhoid fever or pneumonia.

Whiskey is the form oftenest serviceable. It contains about 40 to 45 per cent. by weight of alcohol. Brandy may be employed in equal dose. Port wine is excellent, and is well liked by many children. Its dose is somewhat over twice that of whiskey. Other wines are not so often of value in early life on account of their greater bulk. On occasions, however, champagne is useful for older children. It must be borne in mind that many of the liquid beef preparations on the market are quite strong in alcohol, although their nutritive value is slight, and that there is danger of giving them in excess. They are often excellent as stimulants, as their taste is pleasant. The dose may be determined by comparing their alcoholic strength (p. 167) with that of whiskey.

The amount of whiskey to be given varies with the demands of the case. An average dose every 3 or 4 hours up to the age of 3 months would be 5 to 10 minims (0.31 to 0.62). This may be readily increased during a short period in emergency. The dose at 1 year would be 10 to 20 minims (0.62 to 1.23).

Alcoholic stimulants should be given well diluted. They may sometimes be mixed with the milk-food of the infant; but, if it is important to insure that the full amount be taken, it is better to give them with a little sweetened water from a spoon, or to add them to only so much of the food as is certain of being ingested.

In view of the extensive controversy which has taken place with regard to the employment of alcohol as a therapeutic agent, it would seem to be a fair statement to say that alcohol is a *drug*, which, like all other drugs of power, is capable of doing harm. That it has been greatly abused and

has done much damage in the past through the too free administration of it by physicians is beyond question. That it further is not needed in many cases where it formerly was given unhesitatingly is equally true. That it is able to do much good in certain conditions seems so well supported by the clinical experience of centuries that it is difficult to controvert this by the apparent results of physiological experiments.

Alkalies are well borne and are of particular service in early life, on account of their power to neutralize gastric acidity and restrain vomiting, and for other reasons. Liquor calcis in teaspoonful doses may be given hourly if needed in early infancy, or bicarbonate of soda in doses of 1 to 2 grains (0.065 to 0.13) in the first months of life.

Alkaline salts, such as the citrate of potash and acetate of ammonia, are much employed as febrifuges. Their value for this purpose is somewhat uncertain. 1 to 2 grains (0.065 to 0.13) of the citrate or 15 minims (0.92) of the liquor potassii citratis may be administered every 3 hours at the age of 1 year. Very much larger doses are needed when it is required to render the urine distinctly alkaline.

Antimony is not at all well borne in infancy and early childhood. Its action is often very depressing, whether used as an emetic or as an expectorant. Since other less dangerous drugs readily replace it, it should not be employed. It is mentioned here only to condemn it.

Antipyretic drugs of the coal-tar series are in my experience well tolerated by children, but not often needed to reduce fever, external measures for this purpose being preferable. They are serviceable, however, when such measures are not effective or well borne or for other reasons cannot be employed. In this connection a warning must be uttered against the effort to reduce a child's temperature merely because it is elevated. The temperature rises from very slight causes in early life, and runs correspondingly higher than in adults. Even high fever, unless prolonged, is borne remarkably well in infancy and childhood. Unless it is approaching a dangerous hyperpyrexia, or is attended by threatening symptoms, acute elevation of temperature generally need cause no alarm. When, however, very prominent nervous symptoms attend the fever, such as decided delirium, great restlessness, and the evidence of impending convulsions, antipyresis of some sort is required. It is particularly in these conditions that the coal-tar antipyretics do good by their action upon the nerve centers. Under such circumstances, and if hydrotherapy has been ineffectual, antipyrine and phenacetin are to be depended upon, and are safe in proper doses. Acetanilid is more dangerous. To a child of 1 year $\frac{1}{2}$ to 1 grain (0.032 to 0.065) of antipyrine or phenacetin may be given, and repeated in an hour if necessary. An amount should never be used which will produce a rapid and very decided fall of temperature with much sweating, since the danger from antipyretics lies in this critical fall.

The coal-tar antipyretics are often very serviceable in controlling nervous symptoms of any sort even in the absence of fever; such, for instance, as spasmodic affections, chorea, asthma, and especially pertussis. In the treatment of the last-mentioned antipyrin is one of the best of remedies. For the control of this, as for the management of many nervous disorders, the dose must be larger, and can safely be made so, since there is no danger of critical fall of temperature. An infant of 3 to 6 months may receive $\frac{1}{4}$ grain (0.016) of antipyrine every 3 hours for an initial dose, and one of 1 year 1 grain (0.065). In each case the dose may be increased two- or three-fold, if it is well borne and necessity demands it.

Apomorphia is too depressing a drug to be used in infancy, and even in childhood it may well be replaced by other expectorants.

Aromatic oils, generally in the form of aromatic waters, are particularly useful, well tolerated, and agreeable. Peppermint water is sometimes rather strong for the taste in the first few months of life, and spearmint, fennel (*aqua fœniculi*), or cinnamon water is then to be preferred.

Arsenic is borne well by children. Infants of a year may take $\frac{1}{4}$ minim (0.015) and even up to 2 minims (0.123) of Fowler's solution, well diluted, 3 times a day, if found to agree. In later childhood the dose is often as large as or larger than that for adult life. It is used chiefly for chorea, syphilis, and in some forms of anemia, and has been recommended, too, for chronic gastritis in infancy.

Belladonna is borne exceptionally well in early life. One minim (0.062) of the tincture (equivalent to about $\frac{1}{3000}$ grain (0.000021) of atropine), given 3 to 6 times in 24 hours, is often well tolerated in the 1st year of life. During childhood the drug is often borne in larger doses than in adult life. Yet the individual susceptibility varies extremely, and very small amounts often flush children decidedly. The initial dose should, therefore, always be small, and the attendant informed of the possible result. The variation in the strength among different samples of the tincture render a fresh solution of the alkaloid preferable. The drug is particularly useful in enuresis and in some forms of bronchopneumonia.

Bromides are tolerated unusually well, and are of great service in nervous conditions, especially in convulsions. They are useful also for insomnia and colic. A child of 2 years requires 3 to 5 grains (0.194 to 0.324) at a dose. An infant up to the age of 3 months takes $\frac{1}{2}$ to 1 grain (0.032 to 0.065), but in serious conditions, such as convulsions, 4 or 5 grains (0.259 to 0.324) are required, either by the mouth or be enema.

A pustular eruption is sometimes soon produced by the use of the bromides. In my own experience this is rare, but the possibility of the occurrence must not be forgotten. (See Fig. 422, Vol. II, p. 549.)

Carbolic acid is only to be mentioned on account of the caution to be used in even its local application in infancy. Cases of poisoning have repeatedly occurred, and I have seen severe injury to the skin produced by the local employment of some of the proprietary preparations containing the drug. It is better to avoid it unless specially indicated.

Chloral is generally borne well by infants, but, as it is a powerful narcotic, its action must be watched carefully. It is a valuable remedy in severe colic, although it is occasionally irritating to the stomach. Its especial place is in the treatment of convulsions. The dose is $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.016 to 0.032) for milder ailments up to the age of 3 months. For convulsions at this age 1 to 2 grains (0.065 to 0.13) may be given by the rectum, and repeated as required.

Cocaine is a dangerous remedy in early life, and can well be dispensed with. It is mentioned only to emphasize this fact.

Cod liver oil constitutes one of the very best of the tonic remedies. Children generally take and digest it well. From $\frac{1}{4}$ to $\frac{1}{2}$ fluidram (0.92 to 1.85) in emulsion or in a syrupy malt-extract should be given to children in the 1st year, and double this after this age. It should not be administered where there is gastric indigestion especially of fat, and occasionally it is not well tolerated when there is diarrhea. During very hot weather it is not always well borne, and it may be found necessary to intermit its administration at this time. Its use by intunction, while possibly occasionally of benefit, is unpleasant and generally unsatisfactory.

Digitalis is, in my experience, a useful drug in infancy and childhood, and generally well borne. In the first 6 months the dose of the tincture should be $\frac{1}{4}$ to 1 minim (0.015 to 0.062) repeated as needed. The effect upon the pulse must always be watched with care, and the drug stopped if irregularity is produced. The tendency of digitalis to disagree with the stomach is also to be remembered. The relative value of the newer preparations of digitalis is still under discussion. Digalen, digipuratum, digitalone, digitalin, and others are well spoken of. For the present my preference is still for the tincture, except for hypodermic administration.

Iron is a very useful tonic when there is anemia. It is not needed in other conditions with debility. To infants it should be given in a form as unirritating as possible, such as the lactate, citrate, malate, or pyrophosphate. For older children Bland's pill (*pilulæ ferri carbonatis*); or Vallet's mass (*massa ferri carbonatis*) are excellent. Some of the newer organic compounds on the market are very serviceable, but appear no better than, if equal to, the older official preparations. The possibility of iron producing indigestion is not to be forgotten.

Ipecacuanha is generally remarkably well tolerated. A child of 3 or 4 years, or even younger, can ordinarily take as much as, or more than, an adult. As an expectorant the syrup may be employed in doses of from 3 to 4 minims (0.185 to 0.246) in the first 3 months, and 6 to 8 minims (0.37 to 0.493) at 1 year, every 2 or 3 hours. Yet individual susceptibility exists, and the mother should be warned of the possibility of vomiting being produced. Ipecacuanha is one of the most serviceable and least depressing of emetics. A fluidram (3.7) of the syrup may be given at a dose, and repeated in 20 minutes if there has been no action.

Mercury in nearly all forms is well tolerated by children and seldom salivates. For its constitutional effect in infancy it is conveniently applied in the form of the ointment or the oleate smeared on the binder. Internally for the same purpose calomel may be given in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.016 to 0.032) or gray powder in doses of 1 grain (0.065) or even 2 grains (0.13) 3 times a day to infants under 3 months. The bichloride of mercury, formerly much used in diphtheria, was surprisingly well tolerated. An amount equaling $\frac{1}{32}$ grain (0.002) every 2 hours to a child of 2 years was a common dosage. As a rule, however, the milder mercurials are to be preferred. Bichloride of mercury used locally has produced toxic symptoms in infancy. The action of calomel as a laxative is referred to under Purgatives.

Nitro-glycerine fills a valuable place in the therapeutics of infancy and childhood. It is tolerated in proportionately very large amounts. $\frac{1}{500}$ to $\frac{1}{200}$ grain (0.00013 to 0.0003) by the mouth or hypodermically may be given to a child of 6 months, repeated soon, and frequently in larger dose. It is often of great value where there is failure of the heart's force in various diseases, acting probably by the reduction of the arterial pressure.

Opiates.—In the first 2 or 3 months of life opiates must be given *with great caution*, and in very small initial dose until the susceptibility is ascertained. At any period of early life instructions should always be left with the attendants that the dose is to be lessened or the medicine stopped if the effect has been decided. In greatly debilitated conditions, and in impending coma at any period of infancy, opiates are liable to act with unusual power. Except under these circumstances, the susceptibility diminishes after the age of 3 months, although still present to a

moderate extent. As the individual reaction varies greatly, after the initial tentative dose the amount can be increased until the desired effect is produced. Deodorized tincture, paregoric, Dover's powder, morphine, heroin and codeine are perhaps the most useful preparations. For infants we can well do without the stronger preparations except for hypodermic medication. The average *initial* dose, except in the extremely sensitive states mentioned, may be as follows: Up to the age of 3 months: Deodorized tincture $\frac{1}{30}$ to $\frac{1}{15}$ minim (0.002 to 0.004), camphorated tincture (paregoric) 1 minim (0.062), Dover's powder $\frac{1}{30}$ to $\frac{1}{20}$ grain (0.002 to 0.003), morphine and heroin $\frac{1}{800}$ to $\frac{1}{500}$ (0.00008 to 0.00013), codeine $\frac{1}{300}$ to $\frac{1}{200}$ grain (0.0002 to 0.0003). At 1 year the initial doses may be: Deodorized tincture $\frac{1}{4}$ to $\frac{1}{2}$ minim (0.015 to 0.031), camphorated tincture 4 to 10 minims (0.246 to 0.616), Dover's powder $\frac{1}{8}$ to $\frac{1}{2}$ grain (0.008 to 0.032), morphine and heroin $\frac{1}{150}$ to $\frac{1}{100}$ grain (0.0004 to 0.0006), codeine $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.0006 to 0.0013). Nothing that has been said militates in any way against the proper employment of opiates when needed. At any period of life they constitute in many diseases one of the most valuable of remedies, and the physician who through unwarranted fear avoids their administration to infants or children is separating himself from one of the most useful articles of the pharmacopea.

Purgatives are generally well borne by children, and in relatively larger doses than in later life. Calomel is one of those most frequently employed, although, in my opinion, not in general as serviceable as castor oil, and given too indiscriminately. The dose is proportionately much larger than for adults. Up to the age of 3 months $\frac{1}{24}$ to $\frac{1}{12}$ grain (0.0027 to 0.0054) may be given hourly until $\frac{1}{2}$ grain (0.032) is taken unless an effect is produced sooner. After this age $\frac{1}{10}$ to $\frac{1}{8}$ grain (0.0065 to 0.008) may be given hourly until there is an effect upon the bowels or until 1 grain (0.065) is consumed.

Castor oil is a deservedly popular purgative in infancy and childhood. The dose is proportionately large. Up to 3 months of age $\frac{1}{2}$ fluidram (1.85) may be given, and 1 fluidram (3.7) after this period. Should it tend to gripe, the addition of a small amount of deodorized tincture of opium or paregoric is serviceable. An old-time favorite and a useful mixture consists of equal parts of aromatic syrup of rhubarb and castor oil. Magnesia is also a serviceable purgative. Magnesium oxide (calcined magnesia) usually agrees well with the stomach, but is unpleasant to take. Children of a year may have from 5 to 20 grains (0.324 to 1.3) or more. Magma magnesiae (milk of magnesia), which consists merely of a suspension of magnesium hydroxide in water, is almost a household remedy. Doses of from $\frac{1}{2}$ to 1 fluidram (1.85 to 3.7) may be given in the bottle of milk-mixture in the case of infants artificially fed. Solution of citrate of magnesia is well tolerated and often liked by older children, particularly if it is not too acid in taste. 1 to 4 fluidrams (3.7 to 14.8) or more is the dose for an infant of 1 year; 2 to 4 fl. oz. (59 to 118) at the age of 2 to 4 years or older. The addition of orange juice makes it agreeable to those accustomed to this fruit. Senna in the form of the syrup, and rhubarb in that of the aromatic syrup, are each useful and of agreeable taste. Cascara in some of its more pleasant forms is very serviceable as a regulator of the bowels, alone or combined with phenolphthalein.

Pilocarpine is not at all well tolerated by infants, and should not be used. It is liable to produce dangerous depression.

Quinine is borne in exceptionally large amount. It may be given disguised by syrup of yerba santa or syrup of chocolate as already stated. Quinine chocolates also are often useful, although less trustworthy; and aristochin is a nearly tasteless form of quinine and is often effective. The dose of sulphate of quinine for a child of 1 year may be $\frac{1}{2}$ to 1 grain (0.032 to 0.065) 3 times a day. In cases of malaria larger amounts are generally needed. The drug may be given in the form of suppositories in double the dose mentioned, but the results are less satisfactory.

Salicylic acid and its compounds are well tolerated, except for their tendency to produce vomiting.

Strychnine is an extremely useful drug in early life. It may be given in relatively large doses, if there are no contra-indications. It is, however, in my opinion, often administered with little discrimination where other powerful stimulants are more serviceable. It is quite capable of producing or increasing in children a state of nervous excitability, and it should be avoided whenever such a condition or the possibility of the occurrence of a convulsion is present. At 1 year of age $\frac{1}{100}$ to $\frac{1}{150}$ grain (0.0004 to 0.0006) may be administered *daily* in divided doses, but sometimes larger amounts are required. Older children may well take it in the form of minute gelatin-covered pills. 1 minim (0.062) of tincture of nux vomica contains about $\frac{1}{400}$ grain (0.00016) of the combined alkaloïds, chiefly strychnine. The tincture forms one of the best of tonics in early life.

4. Drugs Grouped According to Their Action.—The following statements may be made in addition to and as a summary of what has just been said, grouping the drugs together according to their action.

(a) **Antipyretics.**—In cases of moderate fever the alkaline diuretics such as potassium citrate and ammonium acetate are very commonly employed, alone or combined with sweet spirits of nitre. The actual value of these drugs in reducing fever has been much disputed. In all more urgent cases, where external measures cannot be employed or are unavailing, the coal-tar derivatives are very useful, especially if nervous symptoms are present. There has been an unwarranted prejudice against them. Many of them are entirely safe if properly used.

(b) **Astringents.**—Bismuth subcarbonate is probably the best of these, alone or in combination with a tannic acid preparation, especially such as tannigen or tannalbin. Full doses should be given, as they are harmless. Opium should not be employed except when fever has gone and the intestinal peristalsis is too active, with a decided loss of liquid from the bowel; or in the dysenteric cases where there is excessive straining.

(c) **Purgatives.**—These are given under the same conditions as in adult life. Castor oil is perhaps the best in infancy, or milk of magnesia for less decided action. In older children citrate of magnesia is one of the best purgatives when taken readily. When a small dose is required, sulphate of magnesia, combined with rhubarb or senna, is efficacious; while cascara, phenolphthalein, and petrolatum liquidum are among the most serviceable regulators of the action of the bowels when a tendency to chronic constipation exists.

(d) **Sedatives.**—The bromides constitute one of the most useful and least harmful drugs of this class for conditions of moderate nervous excitability, sleeplessness and the like; and are frequently serviceable additions to preparations intended to relieve ordinary cough, pertussis, vesical irritability, colic, vomiting in infancy, and threatening convulsions. For the actual attack of convulsions full doses are required, and chloral had

better be used as well. Chloral is serviceable, too, in cases of severe colic in infancy, and occasionally in very decided insomnia. Antipyrine and phenacetin are valuable in convulsive conditions of any sort, and in other nervous states; especially so when fever is also present, but often, too, during apyrexia. They may be employed with advantage to relieve insomnia and headache, and neuralgic pain in general if not too severe. Opium should be reserved for cases of severe pain of any nature, and to relieve harassing cough. It is of value in pneumonia when cough disturbs the rest too greatly, but is to be avoided when respiration is much interfered with, or when abdominal distention is present. It finds a useful place also as a sedative in diseases of the heart.

(e) **Stimulants.**—The indications and contra-indications have already been referred to (p. 223), and will be discussed later in considering the individual diseases in which they may be employed. When a rapid cardiac stimulant is required, as in threatened cardiac failure, caffeine, camphor and adrenalin given hypodermically are to be selected. Strychnine is much employed under much the same conditions, but is, I believe, of less value.

(f) **Tonics.**—Nux vomica is probably the best for use in childhood, especially in cases of loss of appetite and in persistent debility from any cause. It should be avoided when a condition of nervous excitability is present. Iron is useful only in cases of anemia. The action of quinine as a tonic is very variable. In some children it works well. Cod liver oil is one of the best of tonics for chronic states of debilitated health and poor nutrition, if the digestion is in good order.

5. Approximate Average Dosage of Different Drugs.—From what has been already stated the impossibility is manifest of constructing a table of doses for children which can be depended upon in more than a general way. Every medicine must be tried tentatively with every child needing it, and the amount to be given must vary not only with the susceptibility but with the necessity. Urgent cases need vigorous treatment. The following table of approximate dosage for a child of 1 year of age may, however, be of service as a partial guide. The doses in metric equivalents are enclosed in parentheses.

TABLE 67.—TABLE OF DOSAGE AT THE AGE OF 1 YEAR

Acetanilid.....	$\frac{1}{4}$ – $\frac{1}{2}$ gr. (0.016–0.032)
Aconite, tincture.....	$\frac{1}{8}$ – $\frac{1}{4}$ m. (0.008–0.016)
Adrenalin (1:1000 solution).....	2–8 m. (0.123–0.493)
Alcohol.....	brandy..... 5–30 m. (0.31–1.86)
	champagne..... 1–3 dr. (3.7–11.1)
	gin..... 10–60 m. (0.62–3.7)
	port wine..... 10–60 m. (0.62–3.7)
	sherry..... 10–60 m. (0.62–3.7)
Ammonia.....	whiskey..... 5–30 m. (0.31–1.85)
	acetate, liquor..... 15 m. (0.92)
	aromatic spirits..... 2–5 m. (0.123–0.31)
	carbonate..... $\frac{1}{2}$ –1 gr. (0.032–0.065)
Antipyrine.....	chloride..... 1–2 gr. (0.065–0.13)
 $\frac{1}{4}$ –1 gr. (0.016–0.065)
Arsenic, Fowler's solution.....	$\frac{1}{4}$ –1 m. (0.015–0.062)
Asafetida, milk.....	15–60 m. (0.92–3.7)
Atropine.....	$\frac{1}{2000}$ – $\frac{1}{1000}$ gr. (0.000032–0.000065)
Pelladonna, tincture.....	$\frac{1}{2}$ –2 m. (0.031–0.123)
Benzoic acid.....	$\frac{1}{2}$ –1 gr. (0.032–0.065)
	salicylate..... 1–2 gr. (0.065–0.13)
	sul carbonate..... 5–8 gr. (0.324–0.518)
Bismuth.....	subgallate..... 2–4 gr. (0.13–0.259)
	subnitrate..... 5–8 gr. (0.324–0.518)

TABLE 67.—TABLE OF DOSAGE AT THE AGE OF 1 YEAR (*Continued*)

Brandy (See Alcohol)	
Bromides, sodium and potassium.....	1-4 gr. (0.65-0.259)
Bromoforn.....	1-2 m. (0.062-0.123)
Caffeine citrate.....	$\frac{1}{4}$ - $\frac{1}{2}$ gr. (0.016-0.032)
Caffeine sodio-benzoate (hypodermically) ...	$\frac{1}{4}$ - $\frac{1}{2}$ gr. (0.016-0.032)
Calcium chloride.....	1-2 gr. (0.065-0.13)
Calomel (See Mercury)	
Camphor (hypodermically in oil).....	$\frac{1}{3}$ - $\frac{1}{2}$ gr. (0.013-0.032)
Cascara, fluid extract.....	1-4 m. (0.062-0.246)
Castor oil.....	30-60 m. (1.85-3.7)
Chalk-mixture.....	15-60 m. (0.92-3.7)
Champagne (See Alcohol)	
Chloral hydrate.....	$\frac{1}{2}$ -2 gr. (0.032-0.13)
Chloroform, spirits.....	2 m. (-0.123)
Cinchona, compound tincture.....	5 m. (-0.31)
Cinnamon water.....	30-60 m. (1.85-3.7)
Codeine (See Opium)	
Cod liver oil.....	15-60 m. (0.92-3.7)
Corrosive sublimate (See Mercury)	
Digitalis, infusion.....	5-30 m. (0.31-1.85)
Digitalis, tincture.....	$\frac{1}{4}$ -2 m. (0.015-0.123)
Ergot, fluid extract.....	3-5 m. (0.185-0.31)
Fennel water.....	30-60 m. (1.85-3.7)
Gin (See Alcohol)	
Ginger, tincture.....	1-5 m. (0.062-0.31)
Heroin (See Opium)	
Hexamethylenamine.....	$\frac{1}{4}$ - $\frac{1}{2}$ gr. (0.016-0.032)
Hydrochloric acid, dilute.....	1-2 m. (0.062-0.123)
Iodine (See Potash)	
Ipecacuanha. { powdered.....	$\frac{1}{8}$ - $\frac{1}{4}$ gr. (0.008-0.016)
{ syrup.....	3-8 m. (0.185-0.493)
{ syrup (As emetic).....	60 m. (-3.7)
Iron { citrate.....	$\frac{1}{4}$ -1 gr. (0.016-0.065)
{ chloride, tincture.....	$\frac{1}{2}$ -1 m. (0.031-0.062)
{ iodide, syrup.....	1-5 m. (0.062-0.31)
{ lactate.....	$\frac{1}{4}$ -1 gr. (0.016-0.065)
{ pyrophosphate.....	$\frac{1}{4}$ -1 gr. (0.016-0.065)
{ reduced.....	$\frac{1}{4}$ - $\frac{1}{2}$ gr. (0.016-0.032)
Kino, tincture.....	5 m. (0.31)
Krameria, tincture.....	5 m. (0.31)
Lactic acid.....	5 m. (0.31)
Laudanum (See Opium)	
Lime-water.....	$\frac{1}{2}$ -2 dr. (1.85-7.4)
Liquorice-powder.....	2-10 gr. (0.13-0.65)
Magnesia { citrate, solution.....	1-4 dr. (3.7-14.8)
{ milk.....	1-2 dr. (3.7-7.4)
{ oxide.....	5-30 gr. (0.324-1.94)
{ sulphate.....	3-10 gr. (0.194-0.65)
Manna.....	5-30 gr. (0.324-1.94)
Mercury. { calomel.....	$\frac{1}{4}$ -1 gr. (0.016-0.065)
{ calomel, divided doses (as purga- tive).....	$\frac{1}{2}$ -1 gr. (0.032-0.065)
{ grey powder.....	1-2 gr. (0.065-0.13)
Morphine (See Opium)	
Nitre, sweet spirits of.....	2-6 m. (0.123-0.370)
Nux vomica, tincture.....	1-2 m. (0.062-0.123)
Opium .. { codeine.....	$\frac{1}{100}$ - $\frac{1}{50}$ gr. (0.0006-0.0013)
{ deodorized tincture.....	$\frac{1}{4}$ - $\frac{1}{2}$ m. (0.015-0.031)
{ dover's powder.....	$\frac{1}{8}$ - $\frac{1}{2}$ gr. (0.008-0.032)
{ heroin.....	$\frac{1}{150}$ - $\frac{1}{100}$ gr. (0.0004-0.0006)
{ laudanum.....	$\frac{1}{8}$ - $\frac{1}{2}$ m. (0.008-0.031)
{ morphine, sulphate.....	$\frac{1}{150}$ - $\frac{1}{100}$ gr. (0.0004-0.0006)
{ paregoric (camphorated tincture).....	3-10 m. (0.185-0.62)
Pancreatin.....	1-3 gr. (0.065-0.194)
Peppermint-water.....	30-60 m. (1.85-3.7)
Pepsin.....	$\frac{1}{2}$ -2 gr. (0.032-0.13)

TABLE 67.—TABLE OF DOSAGE AT THE AGE OF 1 YEAR (*Concluded*)

Phenacetin.....	$\frac{1}{4}$ -1 gr. (0.016-0.065)
Phenolphthalein.....	$\frac{1}{2}$ -1 gr. (0.032-0.065)
Phosphorus.....	$\frac{1}{500}$ gr. (0.00013)
Potassium { citrate.....	1-2 gr. (0.065-0.13)
chlorate.....	1 gr. (0.065)
iodide.....	$\frac{1}{4}$ -1 gr. (0.016-0.065)
Quinine, sulphate.....	$\frac{1}{2}$ -1 gr. (0.032-0.065)
Rhubarb, aromatic syrup.....	15-30 m. (0.92-1.85)
Salicylic acid (See Soda)	
Salol.....	$\frac{1}{2}$ -1 gr. (0.032-0.065)
Salophene.....	$\frac{1}{2}$ -1 gr. (0.032-0.065)
Santonin.....	$\frac{1}{4}$ - $\frac{1}{2}$ gr. (0.016-0.032)
Senna.....	$\frac{1}{4}$ -4 gr. (0.016-0.259)
Senna, syrup.....	15-30 m. (0.92-1.85)
Silver, nitrate.....	$\frac{1}{120}$ - $\frac{1}{60}$ gr. (0.0005-0.001)
Sodium.. { bicarbonate.....	1-2 gr. (0.065-0.13)
phosphate.....	5-20 gr. (0.324-1.3)
salicylate.....	$\frac{1}{2}$ -2 gr. (0.032-0.13)
sulphate.....	3-10 (0.194-0.65)
Spearmint-water.....	30-60 m. (1.85-3.7)
Squills, syrup.....	3-10 m. (0.185-0.62)
Strophanthus, tincture.....	$\frac{1}{4}$ -2 m. (0.015-0.123)
Strychnine, sulphate.....	$\frac{1}{500}$ - $\frac{1}{200}$ gr. (0.00013-0.0003)
Sulphuric acid, aromatic.....	$\frac{1}{2}$ -4 m. (0.031-0.246)
Tannic acid.....	$\frac{1}{4}$ -4 gr. (0.016-0.259)
Tannigen.....	1-2 gr. (0.065-0.13)
Tannalbin.....	1-2 gr. (0.065-0.13)
Terpene hydrate.....	$\frac{1}{4}$ - $\frac{1}{2}$ gr. (0.016-0.032)
Trional.....	1-3 gr. (0.065-0.194)
Whiskey (See Alcohol)	

B. TREATMENT OTHER THAN BY DRUGS BY THE MOUTH

1. Hypodermic Medication by Drugs.—This holds a minor place in the therapeutics of infancy and childhood on account of the pain and fright which attends the proceeding. It is, however, too much neglected, since under many circumstances it is invaluable; as when a rapid absorption of a medicine is required, where the rebellion against swallowing medicine is excessive, or where it is important that the stomach be spared as far as possible. In cardiac failure medication is far better given in this way, using camphor (1:10 in olive oil), caffeine sodio-benzoate, preparations of digitalis, strychnine, nitroglycerine and adrenalin. In respiratory failure hypodermic injections of atropine are largely employed. Morphine hypodermically may arrest convulsions or exhausting diarrhea. As in the case of adults a hypodermic dose should be, as a rule, considerably less than that given by the mouth.

2. Hypodermoclysis.—The injection of large amounts of sterilized normal saline solution (0.9 per cent.) under the skin, is invaluable in some states of exhaustion or where there has been great loss of fluid from the tissues as a result of diarrhea, vomiting or hemorrhage. Distilled water should be employed in making the solution. The fluid is contained in a glass funnel or in a bottle or other vessel with an opening at the bottom (Fig. 33). To this a rubber tube is attached with a hypodermic needle of fairly large calibre on the other end. All should be carefully sterilized before using. The needle may be introduced into any region with abundant loose areolar tissue, as in the flank, below the scapula, or the lateral parts of the abdomen. The fluid should be warmed before use and either only a small amount poured into the vessel at a time, or the heat maintained by the application of warm cloths or hot water bags about the

vessel (Fig. 35) since from 1 to 2 hours may be required to allow it to enter gradually beneath the skin. From 30 to 250 c.c. (1 to 8.5 fl. oz.) in the early weeks of life may be allowed to flow by gravity, the vessel being suspended about 2 feet (61 cm.) above the patient, and the amount given varying with the weight of the infant. The injection may be made once daily, or oftener if need be. Solutions of gelatin have been given in a similar manner in severe cases of hemorrhage. It is necessary, however, to obtain absolute sterilization of the fluid, or severe subcutaneous suppuration may occur. A simple boiling is not sufficient. It should be prepared in an autoclave. A solution of bicarbonate of soda may be given by hypodermoclysis in cases of severe acidosis. Inasmuch as it is

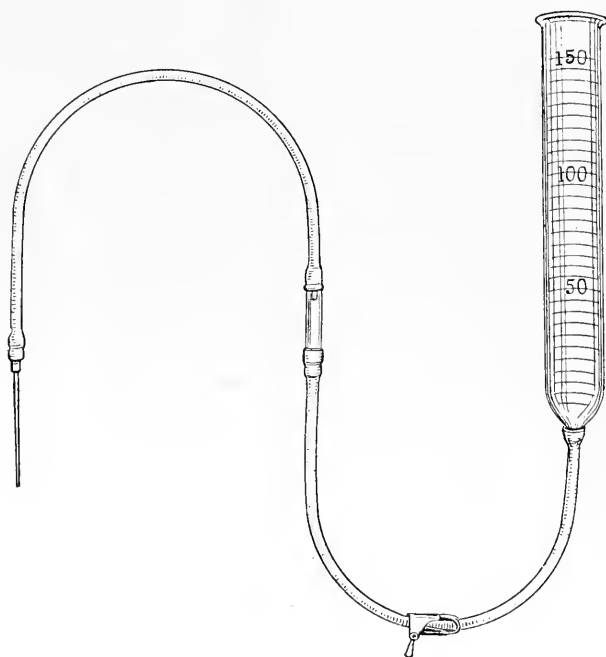


FIG. 33.—APPARATUS FOR HYPODERMOCLYSIS.

The receptacle may be covered with hot cloths if necessary to maintain the heat.

impossible to sterilize this without transforming the bicarbonate into the irritating carbonate, the solution may be prepared as follows: Bring a litre ($33\frac{1}{3}$ fl.oz.) of distilled water to the boiling point. Remove from the flame. Add immediately 30 grains (1.9) of sodium bicarbonate (C. P.) taken directly from the original container and weighed in a sterilized vessel. Cool the solution to a temperature of 110°F . (43.3°C .) and use what is required at once. Even when prepared in this way there is no certainty, however, that it will not prove very irritating, and it is better to administer the solution intravenously. (See p. 245.)

3. Intraperitoneal Injections.—As emphasized by Blackfan and Maxcy,¹ saline solutions may often be advantageously given into the peritoneal cavity instead of by hypodermoclysis. The fluid enters

¹ Amer. Jour. Dis. Child., 1918, XV, 19.

rapidly, and from 100 to 250 c.c. (3.38 to 8.45 fl.oz.) can be introduced in from 15 to 20 minutes. The skin and subcutaneous tissue are picked up between the thumb and finger, and the injection given in the linea alba just below the umbilicus.

4. Suppositories and Enemata.—These constitute a useful method of giving medicines. Quinine may be conveniently administered by suppository, using at least double the dose by the mouth. Suppositories of gluten or of glycerine of small size are serviceable for the relief of constipation, as is often the simple soap-suppository or "soap-stick." Chloral and bromide of potash are to be administered by enema in cases of convulsions, but the size of the injection should not be large or it will not be retained. From 1 to 3 oz. (30 to 89) is sufficient. The enema is conveniently given by the infant-syringe, the fluid being warmed slightly and injected slowly. After the injection the nates must be kept pressed together, or the nurse's thumb held over the anus for a considerable time, to prevent the expulsion of the fluid. Astringent enemata were formerly much employed, as of tannic acid and of nitrate of silver. They must always be weak, especially the latter, or irritation and straining are produced. About $\frac{1}{8}$ grain (0.008) of the silver salt or 1 to 2 grains (0.065 to 0.13) of tannic acid to 1 ounce (30) of water are sufficient, and even this strength is not always well tolerated. The injection of nitrate of silver should be followed by that of a solution of common salt. Suspensions of bismuth are frequently serviceable in cases of irritation of the lower intestinal mucous membrane, using 1 dram (3.9) of the subcarbonate to 4 or 5 oz. (118 or 148) of mucilage of acacia. When medicated enemata are to be employed the bowel should first be unloaded by an ordinary enema. For this purpose, or whenever local treatment of constipation is desired, a small injection of 1 fluidram (3.7) of glycerine, undiluted or mixed with from 1 to 2 oz. (30 to 59) of water often suffices; or a larger one of normal salt-solution or of soapy water may be given, allowing the child to receive as much as it can comfortably hold. When there are hardened fecal masses present an enema of from 1 to 2 oz. (30 to 59) or more of warm cotton-seed oil is very serviceable. This should be retained for some hours, or perhaps over night, and then followed by an injection of soapy water. Nutrient enemata are of very little service in infants, as they are seldom retained if given frequently enough to be of any possible service. They are sometimes of value in older children. The administration of a 5 per cent. solution of glucose in this way has become popular, since it can be absorbed from the large intestine.

5. Irrigation of the Intestine.—In some diseased conditions thorough irrigation of the entire colon is serviceable. The child should be laid on its back on a bed or table, with its hips slightly elevated and its thighs flexed on its abdomen. The bed should be protected by a large rubber sheet, so arranged that it will carry off the water into a receptacle beneath. The injection should be given from a fountain syringe or glass funnel and tube at a distance of about 2 feet (61 cm.) above the child. A hard-rubber nozzle may be used, a small roller-bandage being wrapped about this to act as a plug when pressed against the anus (Fig. 34). A nozzle of this sort allows of the firm pressing of the nates about it, in order to prevent the expelling of the fluid. There is no advantage offered by the employment of the so-called "high injection;" *i.e.* the inserting of an oiled rubber-catheter for a considerable distance into the bowel and the injection of the fluid from a fountain syringe through this. If the fluid of the injection is not expelled promptly, there is only one other direction

in which it can pass, namely, toward the cecum; and it will do this whatever method of administration is followed.

The injection should be given slowly and with occasional cutting off of the water by squeezing or clamping the tube, in order to accustom the bowel to the pressure. There is no fear of over-distention occurring, if the bag of the syringe is not too greatly elevated. The infant will now and then expel the fluid with force at the sides of the nozzle, but the injection should be continued until a gallon (3785) or more has been used, or until the fluid expelled shows little, if any, fecal matter. The colon will hold a surprisingly large amount. A pint (473) may be introduced and retained without distention at the age of 6 months, and 1 to 2 pints

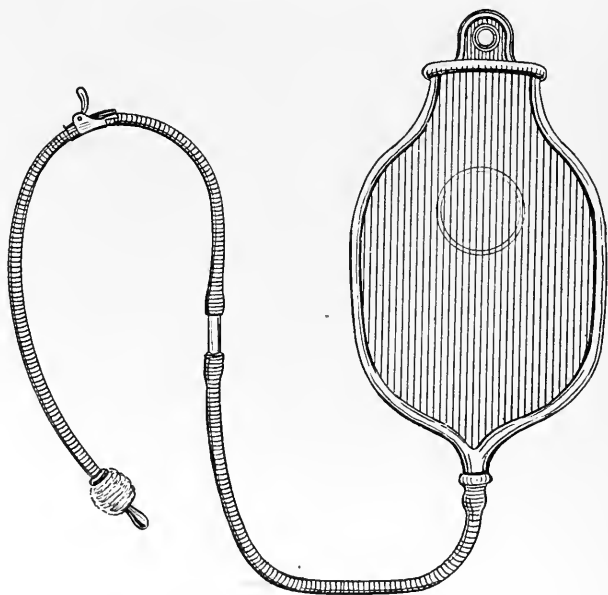


FIG. 34.—FOUNTAIN-SYRINGE FOR INTESTINAL IRRIGATION.

A roller-bandage wrapped around the nozzle checks the expulsion of the liquid.

(473 to 946) or more at the age of 2 years. After the injection is completed the syringe should be removed and the liquid allowed to escape. If a catheter has been employed, it may well be detached from the tube and left in position for a half hour or more until all the water has been expelled, as a portion of it is generally retained for a time.

The temperature of the fluid should generally be about 95° to 100° F. (35° to 37.8°C.) or somewhat less. In some instances slightly cooler injections of 75 to 90° F. (23.9 to 32.2°C.) are better, as where severe local inflammation exists, or where there is high fever, since the reduction of the body-temperature can be accomplished very satisfactorily in this way. Cool injections must not be used in greatly debilitated infants with low body-temperature or where collapse is impending. The fluid employed for irrigation may be simply water, or better, a normal salt-solution (0.9 per cent.) or about 1 teaspoonful to the pint. Where there is much local inflammation irrigation with starch water (1 teaspoonful to the pint, boiled) is often very serviceable. If the starch water is too

thick or grows too cool while passing through the tube, it may jelly and cease flowing. Weak antiseptic solutions are also recommended.

6. Enteroclysis.—In some cases of wasting disease or of collapse a pint (473) or more of warm normal salt-solution may be introduced and allowed to remain (enteroclysis), the syringe being removed and the nates pressed together, the purpose now being to have as much as possible absorbed into the general circulation. A favorite plan is the "drop-method" (Fig. 35), in which the catheter is allowed to remain in place for some hours, with the fluid made to leave it in the form of drops at the rate of about 20 per minute.

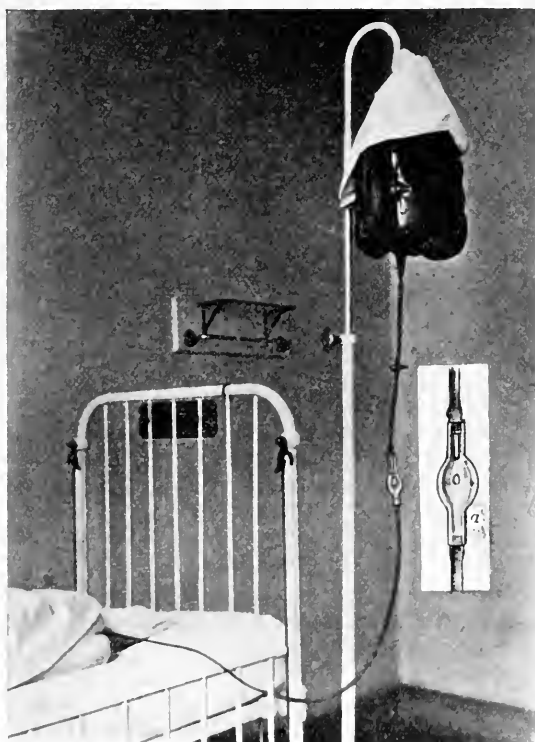


FIG. 35.—APPARATUS FOR THE DROP-METHOD OF ENTEROCLYSIS.

The rapidity of the flow is controlled by a screw-clamp. Hot water-bags surround the vessel containing the liquid. (a) Larger view of the dropper-apparatus.

7. Inhalations.—Inhalations of vapor constitute a most useful plan of treatment in early life. The inhalation may be of water-vapor alone or of chemical substances in the form of a vapor or spray. For the giving of a spray the steam atomizer is very serviceable. The small glass vessel in front of the boiler may be filled with water or lime water, or with solutions containing benzoin, small amounts of turpentine, or other substance as desired. The spray is best given under a croup tent, and should not be allowed to play directly against the face.

A small room may be filled with water-vapor by repeatedly plunging red-hot iron—such as flat-irons, stove lids and the like—or hot bricks or

stones into tubs containing only small amounts of water. The slaking of lime in the room is an efficient old-time method for accomplishing the same purpose. It is impossible, however, to keep a large room satisfactorily filled with steam in this way, and the use of a croup tent around the child is strongly to be recommended (Fig. 36). This may be improvised by placing a large opened umbrella over the patient and draping blankets over this. A better plan is to fasten poles, such as broom handles, upright at each corner of the crib, and to connect the tops of these by cord. Blankets may now be thrown over the framework thus constructed, so that they fall down well about it. An "A" shaped



FIG. 36.—CROUP-TENT.
Steam-atomizer on the table.

opening is left near the head of the patient, and through this the vapor from a croup kettle (Fig. 37) is conducted. The end of the spout should be close to or just within the door of the tent, but not close enough to give too great heat or to permit the child to strike it. The croup kettle is sold provided with an alcohol lamp, and many accidents have occurred through this catching fire, or from the covers of the croup tent coming in contact with it when these have been made of inflammable material. On this account sheets should not be used to form the covering, and, when possible, the croup-kettle should be heated by a small gas stove or electric stove instead of by burning alcohol.

Volatile substances such as benzoïn, turpentine, eucalyptus, etc., may be added to the water in the kettle before the lamp is lighted; or a small sponge may be saturated with them and placed in the enlargement of

the spout. Carbolic acid should be avoided, and creosote used with caution in the case of infants and young children.

Certain drugs may be given in volatile form without the use of water. Thus calomel may be volatilized by pouring it upon a piece of iron heated by a flame.

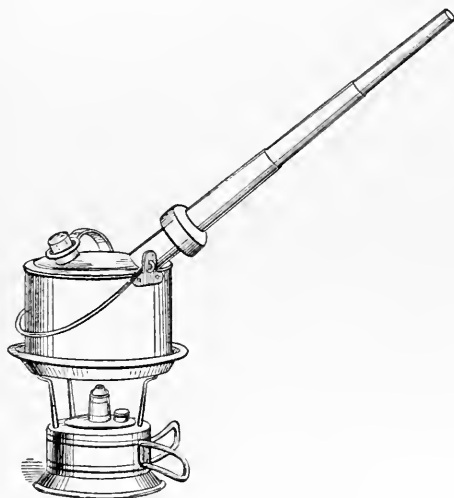


FIG. 37.—CROUP-KETTLE.

8. Inunctions.—Apart from the use of ointments for the local treatment of cutaneous affections, the rubbing with oily substances is sometimes employed to produce general absorption of the drug. Mercury is conveniently given in this way, a small amount of the official ointment being rubbed into the axillæ or groins. A favorite plan in the case of infants is to apply it over the abdomen on a flannel binder. Salicylic acid, especially in the form of salicylate of methyl, is readily absorbed by the skin. Cod liver oil has been much used by inunction. Its odor is extremely unpleasant when employed in this manner and it is questionable whether it offers any advantage over other oils.

9. Applications to the Nose and Throat.—Gargles cannot generally be used before the age of 6 or 7 years, as the child is liable to swallow or reject them. **Painting the throat and nose** evokes resistance in nearly all cases, but is sometimes imperative, and is often easier than the use of the spray. The method of procedure has already been given in describing the examination of the throat.

Substances should be selected which do no harm if swallowed. The application should be made quickly but effectively. This may be done by means of a large camel's-hair brush mounted on a stick, or, better, cotton wrapped firmly upon a stout aluminium applicator. Painting the mucous membrane of the nose with medicated petrolatum is often very serviceable, and is usually a much easier method of treatment than the use of the atomizer unless the patient is docile. A small camel's-hair brush is required, and the procedure should be very gently carried out. In the case of infants the patient may be placed on its back upon the knees of the nurse, with its head hanging somewhat downward, and liquid petrolatum, medicated if desired, may be dropped into

each nostril with a blunt-tipped medicine-dropper. In this position gravity takes the fluid to the upper part of the nostrils.

The hand atomizer may be employed for **spraying the nose or throat**. For oily solutions an apparatus should be chosen which gives a strong spray in a short time and without too much labor. Some are very unsatisfactory. Liquid petrolatum, medicated in various ways as by camphor, menthol, etc., is much used. Aqueous sprays are serviceable for cleansing. For this purpose liquor sodii boratis compositus (Dobell's solution) or other weak alkaline-aromatic solution is serviceable. Peroxide of hydrogen is also much used. A preparation of this should be selected which is not acid, and the dilution should be at least 1:4 for treatment of the nose.

Syringing the nose is sometimes required. For this purpose the best syringe is one entirely of soft rubber (Fig. 38) holding about $1\frac{1}{2}$ fl. oz. (44), or a fountain syringe may be employed with a soft rubber nozzle. The child when debilitated should lie upon the side, the syringe being applied to the upper nostril and the injection continued until the liquid comes clear from the lower nostril. The head and body are then turned to the other side, and the syringing done through the second nostril. The pressure of the water should always be low, lest material be forced into the Eustachian tube and middle ear. In cases where the child is strong it may be allowed to sit, having the head a little forward.



FIG. 38.—OLIVE-TIPPED SOFT RUBBER SYRINGE FOR NASAL DOUCHING.

Syringing the throat is carried out in much the same way, the child sitting upright or lying upon the side. In the case of either the nose or throat, only mild, harmless solutions should be employed, such as normal salt-solution or a weak one of boric acid.

Insufflation of medicine in powdered form upon the mucous membrane of the nose and pharynx finds some place in the therapeutics of children, especially in such diseases as pertussis and diphtheria. The powder may be blown in with an insufflator. The application of drugs in this way to the larynx of infants and young children is in my experience difficult and unsatisfactory; certainly requires special training or skill, and is liable to produce severe laryngeal spasm.

10. Counter-irritants.—Treatment of this sort is particularly valuable in children. The delicacy of the skin in most cases is such that care must be taken to avoid too great irritation.

Blisters are not suitable for infants and not without danger of serious injury to the skin even in childhood.

Mustard plasters or poultices are very serviceable in such conditions as bronchitis. They should be made of mustard with flour or flaxseed meal in proportions depending upon the age. In infancy a strength of 1 of mustard to 5 or 6 of flour is sufficient. For older children 1 of mustard to 3 or 4 of flour will answer. The mustard and flour are mixed dry and then stirred with hot water. The paste is then spread between two layers of thin muslin or linen and applied. The poultice is left on a sufficient time to redden the skin thoroughly, yet not long enough to blister. The time varies with the individual, and the skin should be inspected frequently. The plaster may be applied every day or several times a day according to the effect upon the skin and the requirements of the case. Mustard mixed with white of egg and glycerine instead of

with water is less likely to irritate. The application of mustard in the form of baths or packs is considered later (p. 242).

Pepper plasters and nutmeg plasters were formerly favorites and are sometimes useful. Lard or mutton-suet is spread upon a piece of muslin and liberally dusted with black pepper or powdered nutmeg. The plaster should be worn continuously, until sufficient irritation is produced.

Friction with turpentine and oil, oil of amber, soap liniment, camphorated oil and the like is often of much value in bronchitis, adenitis, and other inflammations. The substance should be rubbed on with the hand or with a small piece of flannel until the skin is slightly reddened. In the case of many infants it seems impossible to produce satisfactory counter-irritation in this way, and the mustard plaster is then to be preferred.

The spice bag is an old-fashioned but very excellent application for colic in early infancy. Equal parts of ground ginger, cloves, cinnamon, and allspice are mixed and put into a small square flannel bag and spread evenly. The bag is then quilted to keep the powder in place. Before applying it should be wet with hot alcohol-and-water or with bathing whiskey. The same bag may be used repeatedly until it begins to lose strength too greatly.

Turpentine stupes are serviceable in abdominal pain or tympanities. A piece of flannel is wrung out in very hot water and sprinkled evenly with turpentine, about half a teaspoonful being used for each square foot of flannel. It is then applied and covered with oiled silk and a dry cloth. Its action must be watched lest too much irritation result.

Dry cups are occasionally useful in cases of severe bronchitis, bronchopneumonia, nephritis or passive congestion of the lungs from cardiac disease. Their action is more powerful than that of friction or mustard plasters.

II. Hydrotherapy.—The employment of water as a remedial measure, applied to the surface of the body in general or to portions of it, is one of the most important of therapeutic agents in infancy and childhood. Yet one of the greatest principles attaching to it is that hydrotherapy is not intended solely or even chiefly for the reduction of temperature. It serves many useful purposes in afebrile states; while when employed for patients with fever its favorable action is to be measured not so much by the degree of the reduction of temperature as by the other good effects produced. Hydrotherapy is often a harmful measure when used, as it too frequently is, with the apparent determination on the part of the physician to reduce the temperature of the body at any cost. Fever is only a symptom, and unless unduly high or prolonged, not one which does harm.

General baths may be divided into (a) sponge baths, (b) tub baths, (c) shower baths, (d) sheet baths, (e) vapor baths, (f) medicated baths. Of the *local baths* may be mentioned (g) foot-baths, (h) compresses and fomentations. Baths may also be classified according to the temperature of the water. A convenient approximate classification is into: (1) Cold bath (40° to 70°F.) (4.4° to 21.1°C.); (2) Cool bath (70° to 80°F.) (21.1° to 26.7°C.); (3) Tepid bath (80° to 90°F.) (26.7° to 32.2°C.); (4) Graduated bath (85° to 90°F.) (29.4° to 32.2°C.) reduced; (5) Warm bath (90° to 100°F.) (32.3 to 37.8°C.); (6) Hot bath (100° to 105°F.) (37.8° to 40.6°C.).

(a) The **sponge bath** with *warm* water should be employed daily in nearly every case of illness. The child should be undressed completely

and laid between blankets. The sponging should be done with care and without exposure, one part of the body being thus washed and dried before another is approached. *Cool* or *tepid* sponging is of great value for the reduction of temperature, and is often in early life as serviceable as is the whole bath in the case of adults. There is not the same need of the precaution alluded to against exposure of the body, since reduction of temperature is the object in view. The sponging should be continued for from 5 to 15 minutes, avoiding the production of decided cyanosis or long-continued depression of the pulse-strength. Some degree of pallor and of lessening of the force of the pulse is a frequent result, and considerable judgment is required to determine whether or not the bath is doing harm. Some children with fever do not bear cool sponging or even tepid sponging at all well.

(b) The **warm tub bath** is an excellent measure in depressed conditions in the eruptive fevers marked by a retrocession of the eruption. Through its action the cardiac strength improves and the rash consequently reappears. It is also a useful antipyretic measure at the beginning of nearly any acute febrile disease, and is often serviceable in reducing temperature in continued fevers when a cooler bath is not well borne. It is a diaphoretic in cases of nephritis; is an excellent sedative in great nervous excitement, insomnia or convulsions; relaxes the spasm of false croup, and reduces the temperature and relieves the dyspnea of bronchopneumonia. Care must be taken to avoid exposure. The room should be warm, the child kept in the bath from 5 to 20 minutes, according to the effect, and wrapped in blankets with little drying as soon as removed from the water. In most cases of fever in infancy and early childhood it is much to be preferred to sponging.

The **hot tub bath** is a powerful stimulant, serviceable in cases of severe exhaustion or collapse, or when the vital powers are failing and atelectasis developing, as in cases of premature birth or of bronchopneumonia in weakly infants. The temperature of the water should be 100°F. (37.8°C.) to not over 105°F., (40.6°C.) *determined by the thermometer*, and the infant should be immersed for not more than 3 minutes. Occasionally the procedure seems to do more harm than good, and to hasten the fatal ending which was inevitable. In other cases, however, the good results are surprising and justify any risk which may attend the measure.

The **tepid tub bath** and the **graduated tub bath** are useful antipyretic measures where sponging or the warm bath fails. In the graduated bath the water is 90°F. (32.2°C.) at the start and is gradually cooled down to 80° (26.7°C.) or sometimes less. Most older children bear bathing of this sort well and the measure is very serviceable in continued fevers, and often fatigues the patient less than sponging. Many children, however, are not benefited, and some do not tolerate baths of this temperature. It is well to bear this in mind and not to persist with bathing merely because fever continues. The child should be in the water from 5 to 10 minutes, being vigorously rubbed meanwhile, and carefully watched against too great depression. In very hot weather, when an infant appears to be exhausted by the continued heat, it is sometimes of advantage to give several tepid tub-baths daily for their bracing and cooling effect.

The **cool and cold tub baths** are not often needed in childhood except in cases of very great hyperpyrexia, such as develops in sunstroke and sometimes in pneumonia and the eruptive fevers. In giving cold baths

under these conditions the temperature of the child must be constantly watched and the bath stopped before apyrexia is reached.

(c) The **shower bath** or **affusion**, is an excellent tonic and stimulant for delicate children. The child should stand in a tub of warm water in a warm room. The cooler water may then be applied from an ordinary shower apparatus, or may be poured over the head and trunk from a pitcher or squeezed from a large sponge. The duration of the affusion should be brief, a minute or less being sufficient, followed by brisk rubbing with a Turkish towel. The water may be tepid, cool, or cold, according to the strength of the child and the degree or reaction which follows. If this latter is not satisfactory, warmer water should be used or the shower bath not employed at all.



FIG. 39.—APPARATUS FOR GIVING A HOT-AIR BATH.

Rubber sheet and outer blanket turned back to show the supporting hoops above the child. Covered metal pipe conducts the hot air beneath the coverings at the foot of the bed.

(d) The **sheet bath**, or **wet pack**, may be either hot or cold. To apply a *cold* pack a rubber cloth is put over the bed and a sheet, previously wrung out in cold water, laid upon it. The child is now stripped, placed upon and enveloped, except the head, in the sheet, and, outside of this, in a blanket. It is often advisable to leave the feet out and to put a hot water bag to them. The child may receive a second pack in 15 or 20 minutes, or may be left in the first for an hour or more if it has fallen asleep. When removed from the pack it should be wrapped in a warm dry blanket.

The cold pack applied in this way is useful for quieting nervousness and often for reducing moderate fever. When a more decided antipyretic action is desired it is necessary to renew the pack every 5 minutes

several times in succession, sometimes using ice water, but only if the temperature of the body is excessively high. It is often more convenient to substitute cloths dipped in ice water for the sheet, since these may more easily be removed, remoistened and replaced. The wrapping in the blanket may be omitted in these cases. A cold cloth must always be kept to the head.

The *hot pack, or blanket bath*, is given by covering the child with towels wrung out in hot water, or wrapping it in a blanket, similarly treated; and then enveloping in several dry blankets. The pack may be renewed in half an hour. The hot pack is often serviceable for producing free perspiration in cases of nephritis.

(e) The **vapor bath** is employed to cause profuse perspiration in nephritis. The bed is well covered by a blanket and the child is stripped and laid upon this. Other blankets are then thrown over the child, reaching nearly or quite to the floor, but kept away from the body by half barrel-hoops, a chair in the bed, or some other support (Fig. 39) except where they are tucked in closely about the neck. Into the air-space thus formed about the child vapor is now conducted from a croup-kettle. The process is continued from 15 to 30 minutes if the child tolerates it well. Care must be taken that the vapor does not play directly against the body, since unconscious children have repeatedly been severely burned in this way. In the absence of a croup-kettle, vapor may be produced beneath the bed by slaking lime, or by hot iron dropped into water, the vapor being given an opening through which it can rise and surround the child.

The *hot air bath* is applied in a similar manner and for the same purpose, dry hot air from an alcohol lamp or small gas-stove being conducted under the covers by a tin pipe (Fig. 39). With either procedure every precaution must be taken against the igniting of the bed clothing.

(f) **Medicated baths** are of much service in childhood. The *warm or hot mustard tub-bath* is a powerful stimulant in cases of cardiac failure or where for any reason it is desired to bring the blood to the surface of the body. Often the good results obtained are surprising. Mustard is added to the water in the proportion of 1 oz. (28) (6 level tablespoonfuls) to 1 gallon (3785) of water. The duration of immersion should be 10 minutes, or less if the skin has become well reddened or if the bath is not well borne. The development of a sensation of tingling produced by the mustard water on the nurse's arms is also an indication to remove the child. The *mustard-pack* is a convenient and useful application in cases of prostration or collapse where the physician fears the greater disturbance of the tub bath. The child is stripped as for the ordinary pack, laid upon a blanket, and covered, except the head, with a cloth dipped in hot mustard water, slightly stronger than that used for the tub bath. The blanket is then wrapped around the body. The pack may continue for 10 minutes or more according to the degree of redness of the skin which it produces. A very efficient method of applying a powerful mustard-pack is recommended by Heubner.¹ I have seen it occasionally produce surprising results in patients apparently almost moribund. Heubner advises the mixing of "2 handfuls of mustard-flour to a liter of warm water, and stirring thoroughly until the odor of the mustard has become distinctly irritating to the nose and eyes. A cloth is now dipped in the mustard water, wrung out slightly, and

¹ Lehrb. d. Kinderh., 1911, II, 269.

wrapped completely about the patient up to the neck, and a blanket wrapped outside of this. The child remains in the pack 10 minutes, and is then washed quickly and thoroughly with warm water, and re-enveloped in a fresh pack of simple warm water, where it remains 2 to 3 hours."

The *starch bath* may be made of the strength of $\frac{1}{2}$ cupful of boiled starch to every 4 gallons (15,142) of water. If the starch has already jellied it may be reheated, or pressed through moistened cheese-cloth. This bath is useful in some affections of the skin. Starch water for washing the skin may be made in the same way.

The *soda bath* consists of a solution of 1 tablespoonful of carbonate of soda (washing soda) to every 4 gallons (15,142) of water. It is used for the same purposes as the starch bath, and is often combined with it by dissolving the soda in the starch water.

Salt baths have been much used as a tonic treatment for debilitated children, particularly those with rickets. Rock salt, coarse table salt, or preferably dried sea-salt may be dissolved in water in the proportion of 4 oz. (113) (10 level tablespoonfuls) to 1 gallon (3785) of water. The child may be washed with or immersed in this after having had the soap suds of the ordinary washing removed with plain water. The duration of immersion depends on the temperature of the water and the condition of the case in general. Doubtless salt baths, if sufficiently cool, do good, but whether this is in any way due to the presence of the salt is questionable.

The *bran bath* is made by putting 1 lb. (454) (about 3 pints) or more of bran into a thin muslin bag and boiling this in water for a quarter of an hour. This water is then added to that of the bath until the whole is slightly milky in appearance. Bran baths have been employed in many irritated conditions of the skin in infants and children.

The *sulphur bath*, used sometimes in chronic rheumatic disorders and in some affections of the skin, is made by dissolving 20 grains (1.3) of potassium sulphide in each gallon (3785) of water employed. It cannot be given in a metal tub.

Disinfecting baths are employed after recovery from infectious diseases. They may consist of a 2 per cent. solution of carbolic acid, for older children or of liquor sodæ chlorinatæ, diluted to the strength of 6 fl. oz. (177) to 1 gallon (3785) of water. A solution of corrosive sublimate of the strength of 1 : 10,000 may be used instead.

Various *mineral springs* furnish whole medicated baths useful in many affections, notably those of Nauheim for the treatment of cardiac diseases, and various hot sulphur springs for rheumatic conditions. Modifications of the Nauheim baths may be given at home.

(g) Of the local baths the *foot-bath* is one of the most serviceable for older children. It is generally given in the form of the hot mustard foot-bath, of the strength of 1 oz. (28) (2 moderately heaping tablespoonfuls) of mustard to 1 gallon (3785) of water. Care must be observed that the room is warm and the bed-clothes also. The child, dressed in its night-clothes, sits on the edge of the bed, well wrapped with blankets, including the thighs, while the feet and legs are in the tub of mustard water. After 5 or 10 minutes the feet are rapidly dried and wrapped in a warmed blanket, and the child put to bed. Exposure is avoided even more completely if the tub is in the bed under the covers, while the child lies with the knees drawn up and the feet in the mustard water.

(h) **Compresses and fomentations** constitute what may be called local forms of baths. *Cold compresses* consist of thin cloth, folded into several layers, dipped in ice water, wrung out and laid on the affected part. They must be light, not wet enough to drip, and changed every few minutes. They are serviceable in inflammation of the eyes, sprains, etc. *Hot compresses, or fomentations*, are made of flannel in several layers, which has been wrung out in water as hot as can be borne. This wringing is conveniently done by dropping the wet flannel into a dry towel and then thoroughly twisting this. The nurse then tests the flannel against her cheek, applies it quickly to the part, and covers it with oil-silk and then with dry flannel or a dry towel. It should be renewed in an hour, or less if it is desired to maintain decided heat.

(i) **Poultices.**—The poultice is intended to furnish a wet dressing which will retain heat longer than a fomentation. It is commonly composed of flaxseed meal, but other substances may be used instead, such as cornmeal, bread, starch, slippery-elm, etc., according to circumstances. The *flaxseed poultice* is made by stirring ground flaxseed into a small quantity of water nearly or quite boiling, until it is of the consistency of hot mush, too thick to flow. This is spread with a case-knife upon a thick piece of cotton or linen cloth, the edges folded over slightly, and the whole covered with cheese-cloth, gauze or a thin old pocket handkerchief. The nurse should test it against her cheek to see that it is not too hot, apply it, cover it with oil-silk or paraffin paper, and enclose with a bandage. It should be renewed every few hours if the heat is to be maintained. The *slippery-elm poultice* and the *cornmeal poultice* are prepared in the same way, from ground elm bark or from cornmeal. They have no special advantages over the flaxseed poultice. The *bread-and-milk poultice* is popular and easily prepared. Stale bread crumb is stirred into hot milk until the proper consistency is attained. It is then spread as described. Any of these poultices are applicable to many cases of local inflammation when there is no open wound. In the latter case an antiseptic fomentation is generally preferred.

In many cases of pain and tenderness, as in some abdominal affections, where a flaxseed poultice would be too heavy, a *bran poultice* may be substituted. A flannel bag is partly filled with bran, thoroughly wet with boiling water, wrung out in a towel, and applied. In place of this a *hop poultice* may be prepared in the same way. Neither of these are as popular as formerly. The *mustard poultice* has been described in considering counter-irritants. The *jacket poultice* and the *cotton jacket*, the latter if properly made acting somewhat like a poultice, are apparently going more and more out of vogue; it seems to me with good reason.

12. Dry Cold.—This may be applied by coils of small lead or rubber tubing, fitted to the affected part, and through which cold water is conducted (Leiter's coils). A more convenient method, however, is the employment of ice-bags. Applied to the head an ice-bag is often of benefit for reducing temperature in febrile diseases; applied over the abdomen it is even more effective for this purpose. It often gives relief from the pain of pleurisy or cardiac disease. Ice is also useful applied below the ears in cases of tonsilitis. The bags employed should, however, be of thin rubber. The long sausage-shaped bag of thick rubber generally sold for application to the neck is not serviceable. The ice-bag used in any region of the body should never be quite filled or it cannot adapt itself well. The local employment of ice must be watched very care-

fully, and is more suitable for older children than for young infants. In the latter it may cause dangerous depression. It is often best to insert one or more layers of thin cloth between the bag and the skin to prevent harmful action upon the latter.

13. Dry Heat.—This is useful particularly for the relief of pain and for the treatment of chronic articular affections. In acute painful conditions, such as otitis; for the relief of shock or collapse; and for low temperature in marantic or premature infants the employment of hot water bags or bottles constitutes an excellent therapeutic procedure. For chronic articular inflammation dry heat may be applied by baking in a special apparatus made for the purpose, or by the use of radiant heat from a group of electric lamps. With all infants and in unconscious states at any age precautions must be carefully taken against burning the patient.

14. Blood-letting.—Venesection is not suitable for infancy and early childhood and only rarely indicated in older children. There are exceptional cases of uremia or of acute distention of the right side of the heart where it does good. Local blood-letting by wet cups should rarely be employed except in strong subjects in later childhood. Leeching may occasionally be serviceable in dilatation of the right side of the heart, uremia, otitis, meningitis, and pneumonia in strong children.

15. Vaccine and Serum Therapy.—This method of treatment has come of recent years into great prominence, but, like all new methods, has been overrated, while possessing undoubted value in some conditions. Vaccine treatment consists in the hypodermic administration of a suspension of dead bacteria. As a prophylactic measure for typhoid fever it appears certainly to have established its position, and for the treatment of the attack it seems to be of value. In most of the streptococcic infections the usefulness of vaccines is doubtful. Sometimes they seem to do good in erysipelas, and the treatment is well worth trying. Staphylococcic infections offer a better field, especially in suppurative processes in the skin. The value of vaccine in pertussis is still under discussion, and it must be stated that the efficacy of the treatment has at least not been proven. The same may be said regarding the employment of gonococcus vaccine in vulvo-vaginitis, and of that of the colon bacillus in pyelitis. Instances are on record of decided apparent benefit; while other investigators have failed to accomplish any good whatever.

The employment of sera, or of the extractive matter from bacteria, is of somewhat older date, but occupies a very limited field of usefulness. The serviceableness of the diphtheria antitoxin is beyond question, as also seems to be that of the serum-treatment for cerebrospinal fever; and that for pneumonia is encouraging. Tuberculin has not maintained the position once hoped for it, and is a remedy by no means without danger. The serum-treatment of scarlet fever has not established itself in the opinion of most physicians, although it may be perhaps of value in the management of the complications. In certain hemorrhagic conditions the injection of a foreign serum, or even of human serum or of blood itself, appears to be distinctly beneficial in controlling the bleeding.

16. Intra-venous Injections.—The employment of this procedure is considerably interfered with in infancy owing to the difficulty in finding a vein. The longitudinal sinus at the position of the fontanelle may be used instead. In older children the procedure is occasionally useful in cases of collapse, as after profuse hemorrhage, using a sterilized warm normal salt-solution (0.9 per cent.). The treatment of syphilis

by arsphenamine is preferably given intravenously. Sodium bicarbonate may be given intravenously in cases of acidosis (see p. 232), or a 5 per cent. solution of dextrose in severe malnutrition in gastrointestinal disorders.

17. Transfusion of Blood.—The introduction of blood from a human subject into the veins of a patient has been found useful in cases of hemorrhage or of severe anemia from other causes. It has also been employed successfully in hemorrhagic diseases in the new-born. The transfusion may be accomplished directly into the vein from the circulation of the individual furnishing the blood, or the blood may be aspirated into a glass syringe and then promptly injected into one of the large veins of the child, preferably the median cephalic or the external jugular.



FIG. 40.—THE PERFORMANCE OF LAVAGE OF THE STOMACH.

Either procedure requires technical skill, the first especially so. The simplest method is the employment of a citrated blood, using 1 part of a sterilized 2.5 per cent. solution of sodium citrate and 4 parts of blood. This prevents coagulation. With any method it is essential first to test the agglutinative action of the blood of the recipient and of the donor upon each other, or dangerous hemolysis may take place (Minot).¹ From 1 to 2½ fl.oz. (30 to 75) of blood may be injected at the age of 6 months or less; 3 to 4 fl.oz. (89 to 118) at 1 year, and 4 to 6 fl.oz. (118 to 177) at 2 or 3 years (Zingher).²

18. Lavage.—Very frequently obstinate acute or chronic vomiting will be made to cease by lavage after the administration of drugs has failed entirely. A stomach-tube is employed, composed of a soft rubber catheter, No. 13 or 14 American scale, connected by a piece of thin glass-

¹ Boston Med. and Surg. Jour., 1916, CLXXIV, 667.

² Med. Rec., 1915, LXXXVII, 440.

tube to a section of soft rubber tubing, the other end of which is attached to a funnel of hard rubber or glass. The infant sits in the lap of the mother while the nurse introduces the end of the catheter moistened with water or glycerin (Fig. 40) into the mouth and gently but quickly passes it backward into the pharynx and downward into the stomach. A slight resistance is felt as the tube reaches the beginning of the esophagus. The finger of the left hand, placed upon the tongue, may be used as a depressor and a guide. There is very little danger of the tube entering the larynx, but should it do so violent coughing and cessation of breathing will inform us of the fact. About 10 inches (25 cm.) of the catheter should be inserted, measuring from the gums. The funnel is then raised slightly to permit gas to escape and the solution is then poured into it and allowed to find its way into the stomach. Pinching the tube between the finger and thumb and sliding these along it toward the mouth is sometimes needed to start the flow.

After from 4 to 8 fl.oz. (118 to 237) have been given the funnel is lowered as far as possible, in order to syphon out the gastric contents into a basin. The tube is then pinched to prevent air from entering, the funnel raised and more fluid poured in. This process is repeated several times until from a pint to a quart (473 to 946) of fluid has been employed and the washings come away clear of curds or mucus.

The solution ordinarily used is a warm (100° to 110°F.) (37.8° to 43.3°C.) normal saline solution (0.9 per cent.), simple boiled water, or, where there is much mucus, an alkaline solution containing 1 per cent. of bicarbonate of soda. The procedure is entirely without danger, very easy to carry out during infancy, and causes, as a rule, little or no exhaustion. It may be performed daily until no longer needed.

19. Gavage.—The forced feeding of infants is so closely connected with lavage that it may well be described in this connection. After the stomach has been washed, the tube may be left in position and the child fed through it. The funnel should be held elevated for a moment before the food is poured into it, in order to permit gases to escape from the stomach. Instead of sitting the child should be flat upon its back, as it is less likely to regurgitate the food when in this position. For the same reason the child must be kept entirely quiet after the gavage is over. In older subjects it may be necessary to use a mouth-gag to prevent the tube from being bitten, or to protect the finger from the child's teeth. When the tube is about to be withdrawn it should be pinched tightly to prevent food from dropping from it into the pharynx, as this is liable to induce vomiting. The last part of the withdrawal should also be done very quickly. Should vomiting occur immediately, a second gavage may sometimes be given at once with advantage.

Gavage is very easy to perform and is extremely useful in the case of infants who for any reason are unwilling or unable to take sufficient nourishment, or in those where vomiting is troublesome. In the latter case lavage should precede each gavage; in the former lavage is not necessary. It is constantly observed that food given by gavage will often be retained much better than when swallowed. Cases where extreme anorexia threatens life may often be fed with success by gavage. The food may sometimes be thoroughly peptonized with advantage. Sometimes it must be weak on account of an impaired digestive power; in other cases, where the trouble is the refusal to take nourishment in the ordinary way, it may often be given rather stronger and in larger amount than usual, since the intervals of feeding are longer. Children may be fed in this

way 3 or 4 times a day, or occasionally more frequently. In some cases where vomiting or lack of power to swallow is not a factor, but where unwillingness to take food is the cause, it is not necessary for the tube actually to enter the esophagus, but a short piece of tubing may be passed through the mouth to the pharynx and the liquid introduced from a funnel or syringe through this.

20. Nasal Feeding.—In older children where the jaws are tightly shut and a mouth-gag would be required to permit of inserting a tube into the esophagus, it is better to feed through the nose. It is also a serviceable method of feeding after intubation. The procedure is similar to that described for gavage, except that a smaller tube is required, No. 8 or 10 American scale (Fig. 41). In some instances it is better to use the nasal tube for the performing of lavage.

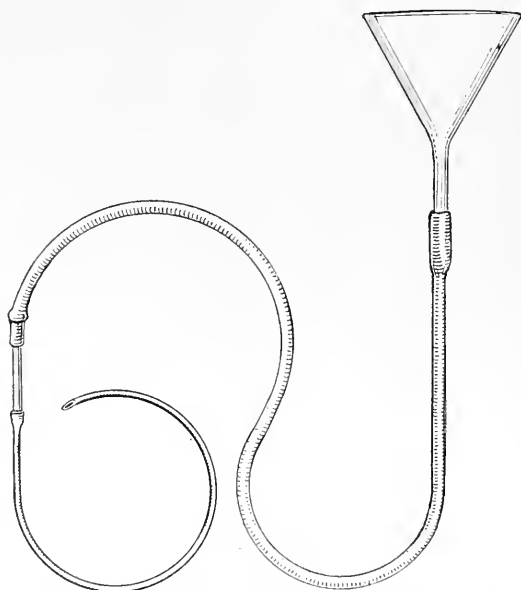


FIG. 41.—FUNNEL AND TUBE FOR NASAL FEEDING.

21. Anesthesia.—Little can be said in this connection which does not apply to adults as well. As a rule anesthetics are well borne in early life. There is a possible danger of sudden death in cases of lymphatism, but this may occur in those of older age as well. Ether is by all odds the safest anesthetic and the one usually to be preferred. The excitement and resistance which it occasions may be avoided by beginning the anesthetization with ethyl chloride, which, however, is a more dangerous anesthetic if depended on solely, particularly in infancy. Nitrous oxide may be used in the same way, or alone for short operations in older children, but is not as safe for infants. Ether is generally to be avoided in diseases of the lungs and in nephritis. Whether ether or chloroform is selected, the vapor should be administered slowly and with the admixture of plenty of air; and this applies with especial force to chloroform.

22. Psychotherapy.—The enormous importance of this form of therapeutics in early life is too frequently overlooked. Even in early

infancy it is seen, for instance, in the good effect of mental quietude or the influence of a calm and somewhat phlegmatic nurse in the controlling of troublesome vomiting; as also in the relief of insomnia by such measures as the avoidance of excitement before the hour for sleep, the removal of noise or light from the room, and the like. In older children the insistence by the physician that the mother shall not speak of nervous symptoms in the presence of the patient, and that these shall be in every way ignored, is something we cannot afford to forget if good results are to be obtained. The remarkable influence of complete change of surroundings and of removal from association with over-anxious parents is shown by the frequent, prompt disappearance of spasmodic symptoms, nervous anorexia or dysphasia, or other nervous manifestations when a child is placed under hospital influences or sent from home in the care of a properly selected nurse. The control of the exhibition of anxiety by the parents and of the bad methods of training shown by them is often the principal and the most difficult object of treatment. The subject is too large for further discussion in this connection. It is referred to briefly to some extent in the chapter upon Diseases of the Nervous System, and I have discussed it also elsewhere.¹

23. Mechanotherapy.—Massage, even when carried out in a very simple and unscientific manner, and in a way which any mother can do, occupies a valuable place in the therapeutics of infancy. The rubbing of the body with the flat of the hand, lubricated with sweet oil or cocoa butter, is an excellent procedure in feeble circulation, malnutrition, anemia, and paralytic conditions. Still better is it to encircle the arm or leg of the infant with the well-greased thumb and forefinger, and to push up and down the limb with the exercise of considerable pressure. More effective is a gentle kneading of the muscles done by a trained masseuse. The procedure usually soon becomes very soothing and agreeable to the patient. Care must always be taken to avoid undue exposure of the body lest chilling result. In older children a more systematic massage, given by one experienced in the method, is often useful, especially so in paralytic or pseudo-paralytic states, faulty habits of sitting or walking, lateral curvature of the spine, and the like. Abdominal massage is frequently one of the best remedies for obstinate chronic constipation. In the case of active children massage does not fill the place which it does with adults, since the child takes relatively far more exercise than the average man undergoes.

Resisted movements may be used either alone or combined with massage. They are serviceable, for instance, in some cases of paralysis and of diseases of the heart and for the training of any especially weak muscles.

For children sufficiently old **gymnastic exercises** are of great value conducted by a trained observer and selected to meet the needs of the individual case. Even without such an advantage, a series of exercises can be chosen by the physician and supervised by an intelligent mother or nurse in the patient's home. These may be used to overcome the results of faulty habits of standing, sitting or walking; distortions of the chest following rachitis or pleurisy, or deformities acquired in other ways. It is necessary, of course, to know the action of the different muscles and to outline special exercises to bring these into play. The reader may be referred to treatises upon exercise and upon orthopedic surgery, and

¹ New York Med. Jour., 1911, June 6.

especially to the interesting contribution by Keating and Young upon *Physical Development*.¹

Rest, with or without isolation, is often most important. Thus in chorea, many cases of cardiac disease, epilepsy, and hysteria it is by far the most important part of the treatment. The fact that nervous, active children are constantly liable to take by far too much exercise is always to be remembered. In such cases systematic enforced rest during a portion of the day is to be enjoined. Many functional nervous disturbances may be aided by the prescribing of recumbent rest for an hour in the middle of the day. That the child sleep at this time is an advantage, but need not be insisted upon. If he is wakeful or restless, he may be allowed to look at a picture-book or to have some not too exciting story read to him.

Electricity is a useful form of mechanotherapy in paralytic conditions and in some other affections. The fright which the application may cause often interferes largely with the benefit which might be expected. Only the weakest current should be employed at first, and never one of sufficient strength to cause pain. Indeed, the first application may well be that of the wet sponges alone, the battery being in action but unconnected. The current to be chosen depends upon the nature of the disease. As a mere matter of muscle-exercise the faradic is indicated.

24. Radiotherapy.—The value of radiotherapy as a therapeutic method has been constantly coming into increasing prominence. Although its field is as yet limited, it has established its value in such conditions as some forms of splenic hypertrophy, notably leukemia; enlargement of the thyroid and thymus glands; cervical and tracheo-bronchial adenitis; and in eczema, tinea tonsurans, nevi, and other affections of the skin.

25. Climatotherapy.—It is a matter of everyday experience that the cure or relief of many diseased conditions will be accomplished more surely and promptly by change of climate than in any other way. Children with subacute bronchitis often react quickly to such a procedure. Asthma, pertussis, chronic or recurring rheumatism, chronic nephritis, tuberculosis, anemia, a debilitated state of the general or the nervous system, and slow convalescence from many acute diseases, are all aided by it. The choice of locality is to be made carefully. A fuller discussion is out of place in this connection, and reference may be made to works upon Climatology. Here it may be said only that many of the good effects of change of climate can be obtained by a more careful regulation of the hygiene at home, particularly in the matter of obtaining sufficient fresh air in the living rooms, both by day and by night, a life largely in the open air, and the guarding against overheating from too great warmth of the clothing.

¹ Keating, *Cyclopedia of the Diseases of Children*, 1890, IV, 301.

DIVISION II

DISEASES

SECTION I

DISEASES OF THE NEW BORN

Monsters and similar malformations arise during *embryonal* life, that is, through the first 2 lunar months. They are the products of defective development rather than of disease and are properly considered in works on teratology rather than on pediatrics. Some of them will be briefly mentioned in other sections. Certain of the diseases of the *fetus*; *i.e.* developing during the last 8 lunar months of intra-uterine life, continue as diseases of the new born, since the pathology is the same for both periods of life. Others, however, seem incompatible with the continuance of life outside the uterus and cannot properly be considered at any length in works treating of the diseases of infants and children. Consequently, any grouping of Diseases of the New born is at best artificial and incomplete, and only to be excused by the greater convenience for study which it offers. In the following pages, therefore, are considered chiefly (1) some of those diseases of fetal life which are capable of continuing, at least at times, in the living child at birth, and (2) certain affections which are peculiarly liable to be acquired by the new-born child or are witnessed only at this time. There are excluded from this section or mentioned only briefly (1) numerous diseases of the new born which are more conveniently treated of later, (2) diseases limited to fetal life and not seen in the living infant; and (3) most instances of defective embryonal development; *i.e.* matters pertaining to malformations and to teratology in general.

CHAPTER I

PREMATURE INFANTS

Etiology.—The frequency of premature birth varies to some extent with the locality and the influence of attendant circumstances. There is a range of from 5 to 25 per cent. according to the statistics of institutions, as published by Rommel.¹ An average might be fairly assumed as 9 per cent. of the total births (Schauta).² After the 38th week of intra-uterine life the infant can no longer be called premature, inasmuch as it is then perfectly developed to all intents. In the absence of actual knowledge of the duration of the pregnancy, the existence of prematurity must be determined according to somewhat arbitrary standards. In general it is assumed that a birth-weight of less than 2500 grams (5.51 lb.) or a length of less than 45 cm. (17.72 inches) is an indication that the child is probably premature. Yet there is a great variation in the weight of premature infants, similar to that seen in those born at full term; and the criterion of length is decidedly more reliable, although even this is not entirely to be depended upon.

A distinction is to be made between cases of congenital asthenia and those of premature birth. The latter, it is true, generally exhibit debility, but not necessarily so; whereas the child with congenital debility born at full term may present many of the evidences of imperfect development characteristic of the prematurely born. The distinction, therefore, cannot always be made clinically with clearness.

Among the causes of premature birth are especially conditions affecting the mother. Syphilis is probably chief of these, but acute infectious disorders, pneumonia, nephritis, tuberculosis and diseases of the heart occupy a prominent etiological position, as do violent exercise and trauma. Twin births are very likely to be premature, Miller³ finding prematurity in 60.6 per cent. of 3380 twin births. These estimates were, however, based upon the size and weight of the infants, which are less applicable to twins than to single births.

Characteristics at Different Periods of Intra-uterine Life.—

A brief description may be given of the characteristics of the infant born prematurely after different periods of intra-uterine life which are compatible with at least temporary viability. The appearances at earlier periods than these have no bearing upon pediatrics. The lengths and weights of the body are those given by Ballentyne.⁴ The months are calendar months:

At 24 weeks (5½ months), the fetus measures from 28 to 34 cm. (11 to 13.4 inches) in length and weighs 676 grams (1.49 lb.). There is a large amount of lanugo, the skin is wrinkled and the vernix caseosa is present. The eyebrows and lashes are evident. The deposit of subcutaneous fat has only just commenced. The testicles have descended to the internal inguinal ring. The eyelids have become separated.

¹ Pfaundler und Schlossmann, *Handb. der Kinderheilk.*, 1906, I, 2, 492.

² Eulenberg's *Real Encyclopädie*, VIII, 120.

³ *Jahrb. f. Kinderheilk.*, 1886, XXV, 181.

⁴ *Antenatal Pathol.*, 1902, 77.

At the close of 28 weeks ($6\frac{1}{2}$ months), the fetus measures about 38 cm. (15 inches) in length and averages a weight of 1170 grams (2.58 lb.). The whole body, except the palms and soles, is covered with lanugo and the vernix caseosa is present. The hair on the head is about 0.5 cm. (0.2 inch) in length. The skin is dull-reddish and somewhat wrinkled. There is little subcutaneous fat. The pupillary membrane which had previously covered the pupil has commenced to disappear. Meconium is found in the intestine. The testicles have nearly or quite descended.

At the age of 32 weeks ($7\frac{1}{2}$ months) of intra-uterine life, the prematurely born child measures from 39 to 41 cm. (15.4 to 16.1 inches) and weighs 1571 grams (3.46 lb.). The lanugo has diminished on the body and the hair increased on the scalp. The skin is still dark-red and wrinkled, although there is more subcutaneous fat than before. The nails are harder and horizontal, but do not project beyond the ends



FIG. 42.—PREMATURE INFANT.

Born at $6\frac{1}{2}$ to 7 months; weight on admission to the Children's Hospital of Philadelphia when 10 days old 2 pounds, 8 ounces (227). The size of the infant can be estimated by comparison with the 8-ounce nursing-bottle.

of the fingers. The pupillary membrane has disappeared. The testicles have fully descended. The infant is very feeble, of low body-temperature, hardly opens the eyes, and cannot suck.

At the age of 36 weeks ($8\frac{1}{2}$ months), the fetus measures 42 to 44 cm. (16.5 to 17.3 inches) and weighs 1942 grams (4.28 lb.). The subcutaneous fat has very decidedly increased and the wrinkling of the skin is much less, while the face is more rounded. The lanugo has largely disappeared. The deep-red color of the skin is now found only on the genitals. The hair on the scalp is over 1 cm. (0.39 inch) in length. The nails do not reach the finger tips. Children born at this period should live under careful attention.

General Symptoms.—The general symptoms characteristic of prematurity vary with the duration of the period of intra-uterine life. In typical cases the cry is feeble and infrequent; the body-temperature is low and maintained at the normal only with difficulty; the infant moves its limbs but little and lies most of the time in an inactive torpid state; there is a great tendency to atelectasis and consequent cyanosis which recur readily after temporary successful treatment; the skin is often icteric and red, or pale, or cyanotic; the respiration is markedly

irregular, feeble and intermittent; the power to suck is often absent, and swallowing is slow. Inasmuch as the infant was born before its natural time, all the organs are in an imperfectly developed state.

Course and Prognosis.—Even with successful treatment the progress is slow; repeated relapses into atelectasis are prone to occur; convulsions and pneumonia are liable to develop; the danger of infection is decided; the digestion is often feeble; a toxic condition often develops; there is constant difficulty in maintaining the body-temperature, and the occurrence of any chilling may be followed by fatal results; the gain in weight is slow; and hemorrhage within the cranium and elsewhere takes place readily. Other things being equal, the chance of living depends upon how early the special care required is commenced, and the skill with which this is conducted. In addition to this, and very largely, the prognosis for final recovery is determined by the intra-uterine age. A child born at the end of 24 weeks usually dies in a few hours. At the close of 28 weeks, recovery occurs in about half of the cases with sufficient care. At the end of the 32d week the majority of the infants will live with proper attention. The statistics of Potel¹ are interesting in this connection. They are shown in the following table:



FIG. 43.—PREMATURE INFANT.

Same case as in Fig. 42. Now aged 6 months. Weight 7 pounds, 10 ounces.

TABLE 68.—VIABILITY OF PREMATURE INFANTS

	Per cent.
56 infants born at 6½ fetal months; 45 died	= 80.4
131 infants born at 7 fetal months; 76 died	= 58.1
53 infants born at 7½ fetal months; 17 died	= 30.1
110 infants born at 8 fetal months; 39 died	= 35.5

Only 1 case survived out of 26 born at the end of the 6th fetal month. A few cases are reported in medical literature of survival at even earlier periods, as, for instance, those recorded by Smyth (21 weeks) and Barker (23 weeks).²

As already stated, the weight alone is not a safe prognostic guide, since it by no means always corresponds with the actual intra-uterine age of the child. Some remarkable instances of recovery are on record in infants with unusually small birth-weights; as, for example, the case reported by Mansell³ in which the baby weighed 18 oz. (510) at birth and survived, and that by Bonnaire⁴ of an infant weighing 820 grams (28.8 oz.). The length of the infant is a better prognostic guide, especially as the intra-uterine age is often difficult to determine with accuracy. Ostréil⁵ gives the following serviceable table:

¹ Thèse de Paris, 1895. Ref. Délestre, Thèse de Paris, 1901.

² Ref. Kleinwachter in Eulenberg's Real Encyclopädie, VIII, 60.

³ Brit. Med. Journ., 1902, I, 773.

⁴ Bull. soc. de méd. légale de France, 1912. Ref. Arch. f. Kinderh., 1913, LIX, 213.

⁵ Monatsschr. f. Geburtsh. und Gynäk., 1905, XXII, 45.

TABLE 69.—VIABILITY OF PREMATURE INFANTS

40 cm. (15.8 inches)	Chances of living	21 per cent.
41 cm. (16.1 inches)	Chances of living	20 "
42 cm. (16.5 inches)	Chances of living	25 "
43 cm. (16.9 inches)	Chances of living	28 "
44 cm. (17.3 inches)	Chances of living	51 "
45 cm. (17.7 inches)	Chances of living	50 "
46 cm. (18.1 inches)	Chances of living	55 "
47 cm. (18.5 inches)	Chances of living	58 "

The large majority of deaths occur in the first weeks. The premature infant, otherwise healthy, which survives will develop as well in later years as the one born at full term.

Treatment.—Among the principal difficulties in the treatment of premature infants are the maintenance of the body-temperature; feeding; the control of imperfect respiration; and the avoidance of infection. For

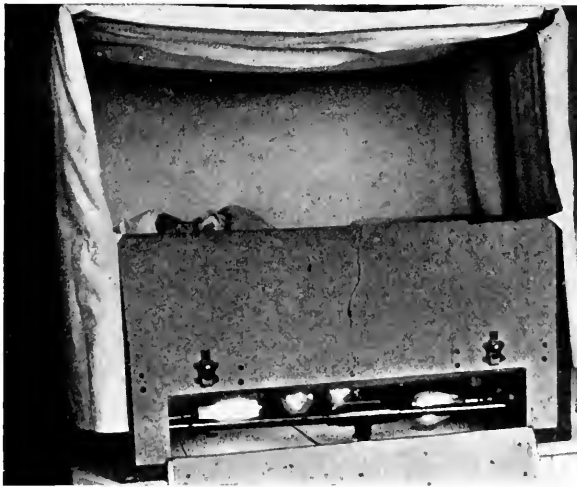


FIG. 44.—BED FOR PREMATURE INFANT.

Iron framework covered with asbestos-board. Cover thrown back. Door open at bottom showing electric lamps.

many years the accomplishing of the first of these was attempted by the employment of an *incubator*, and numerous forms have been devised. In all the effort is made to maintain the temperature automatically at a fixed degree, and to supply abundant warm, fresh, moist air. The difficulties attending this successful ventilation are so great that, on the whole, incubators have been found unsatisfactory even in practise in institutions. A much better plan is to have a room set apart for the purpose, in which the temperature is kept at from 85° to 90° F. (29.4° to 32.2°C.) and fresh warm air constantly supplied.

In institutions where no such room is available, as well as in private practice, very good results may be obtained in other ways, if sufficient intelligent attention is given. A lined clothes-basket or an infant's metal bath-tub may be partially filled with absorbent cotton and the child placed in this after having been oiled and wrapped in warmed cotton and then in blankets. Hot water-bottles are concealed in the cotton in the tub, close to but not touching the infant. A thermometer in the

cotton shows the temperature attained here. It should range from 85° to 95°F. (29.4° to 35°C.), and must be carefully watched, as otherwise the infant's temperature may readily become too high. The bottles should be refilled with hot water as needed, but not all of these at the same time, in order to keep the temperature of the bed as uniform as possible. Electric warming pads are theoretically excellent but practically not without danger, as a short circuit may develop and the bed-clothing be burned. I have, however, used them with decided satisfaction. A very serviceable device consists of a box-like crib of asbestos cardboard or of metal, with an upper and a lower compartment. In the lower a series of electric lamps are installed, and any number desired



FIG. 45.—
BRECK FEEDER.

turned on to produce the required heat in the bed. In the upper compartment is placed a thin mattress or a blanket, and over this cotton, and the infant enveloped as already described. Perforations in the partition between the compartments allows the heated air to rise. A sheet or blanket should be suspended on a support over the upper end of the bed to cut off excessive light and any draughts. I have used a device of this nature with success at the Children's Hospital of Philadelphia (Fig. 44). The room in which the infant is kept should receive an abundance of fresh, clean, moistened, warm air, and the room-temperature maintained at 80°F. (26.7°C.) or sometimes over this. The infant should have no clothing whatever, and the cotton be arranged to cover the whole body except the head. A diaper, or an extra layer of absorbent cotton, should be laid under the nates. In changing this, and, in fact, under all circumstances, the child must be handled or exposed as little as possible. Unless strong enough to nurse from the breast, it is not necessary to remove it from the receptacle even for feeding, but a frequent change of position in bed is important. Bathing should not be employed at all, unless it be hot baths as needed for the relief of atelectasis. Once a day the child may be rubbed all over with warm sweet oil. After an evacuation of the bowels or bladder the nates may be cleansed with cotton and warm oil. The infant's rectal temperature should be taken every few hours, and an effort made to keep it at over 98°F. (36.7°C.). When it is found that the temperature is maintained without artificial heating, this may be gradually abandoned, and the child dressed in the ordinary manner.

The best food by all odds, however, is a suitable breast-milk. In fact this is usually the only food with which satisfactory results can be obtained. The infant may nurse from the breast when it has the power to suck. If it has not, the milk may be pumped from the breast and given in a medicine dropper or the Breck feeder (Fig. 45), the infant not being removed from the bed. It is often best to dilute the milk $\frac{1}{2}$ with water. When there appears to be difficulty in swallowing gavage may be employed. As soon as the infant is able to suck, feeding should be directly from the breast, if possible.

In the cases where substitute feeding is absolutely unavoidable, it is necessary to determine what artificial food will answer best. Its strength in all the elements must be very weak at first, since the power of digestion is but feeble. The direction in which an increase in the strength

of the food shall be made can be determined only by observing the results upon digestion, as shown by the character of the stools, the frequency of vomiting, and the like. According to the individual case we may employ whey-mixtures, buttermilk, food consisting chiefly of dextrin-maltose, or peptonized milk. It would seem best to have the percentage of fat low at first and to increase it with great caution. As soon as needed the strength of the food in general and the amount given may be increased; but with care against over-feeding. The infant should be weighed daily, guarding against chilling by exposure, since it is principally by the weight and the general satisfactory appearance that we can determine that it is thriving. Failure to gain properly may mean that more food is required. Sometimes, if there is indigestion, it may be an indication to reduce the amount.

Regarding the amount of food needed and the frequency of administration, there can be no absolute rule. The caloric needs of the premature infant are probably from 110 to 130 calories per kilogram (50 to 59 per lb.) daily, exceeding those of the full-term child. This is the result of the greater body-surface as compared with the body-weight. This does not apply, however, to the 1st week, and perhaps not until 1 or 2 weeks have passed. Indeed in the first 2 or 3 days we may well be content with a sugar-solution. Benedict and Talbot¹ estimate that the energy requirements of the full-term infant during the first 6 days are only 62 calories per kilogram (28 per lb.). It is probable that the premature infant requires no more or even less than this during this period. The amount of nourishment taken in the early weeks is very small, varying with the weight of the premature infant (125 to 345 c.c. (4.23 to 11.67 fl.oz.) in the first 10 days. Délestre).² The best plan is to calculate the number of calories probably required, and to give a daily amount of food which will supply these; using the calculation, however, only as a guide, and allowing especially the progress of the infant in weight and the condition of its digestion to determine the increase. The frequency of feeding is to be based upon the amount which the infant can take and retain at a time. If it refuses to swallow more than 2 or 3 drams (7 or 11), or vomits if more is forced upon it, feeding every 1 to 2 hours may be necessary. If it can ingest a proper amount at one time, intervals of $2\frac{1}{2}$ to 3 hours will be chosen. In general we may aim for a quantity of from $\frac{1}{2}$ to 2 ounces (15 to 57) every $2\frac{1}{2}$ to 3 hours in the early weeks, depending upon the weight, length, and age of the infant. Vomiting resulting from the administration of too much food is to be avoided as far as possible, on account of the danger of the regurgitated milk entering the respiratory passages and producing asphyxia or aspiration-pneumonia.

The treatment of the premature infant is in other respects symptomatic. The relief of atelectasis and cyanosis is to be obtained by reflex stimulation, as by plunging into a hot bath of 100 to 105°F. (37.8° to 40.6°C.), preferably containing mustard, in order to produce crying. The inhalation of oxygen is often of service, and a retort of the gas should be close at hand for immediate use when required. Infection is to be very carefully avoided, especially by great attention to the care of the navel, and the employment of sterile water for any washing necessary.

¹ Carnegie Instit. Wash., 1915, 233. Ref. Editorial, Journ. Amer. Med. Assoc., 1916, LXVI, 1466.

²Thèse de Paris, 1901.

CHAPTER II

SEPSIS IN THE NEW BORN

The septic infections of the new born include a number of conditions which clearly are due to the entrance of pyogenic germs into the organism. Many of these exhibit also such special peculiarities or localization that they may be described as distinct diseases. Still others are possibly septic, yet are of an origin not clearly understood. General septic infection as it occurs in the new born is the topic now under consideration.

Etiology and Pathological Anatomy.—Sepsis is still too common, but its frequency is trifling as compared with the former extreme prevalence of the affection. In rare instances septic infection may take place before birth. In this event the fetus generally is born dead. The cause in such cases may be a penetration of bacteria or of toxic substances from the mother through the placenta, the mother herself being ill, often with sepsis. In some instances the infection is through the amniotic fluid, in not a small number of cases depending upon a maternal perityphilitic inflammation (Hellendall).¹ In sepsis acquired in this way the primary lesion is a pneumonia. More often sepsis develops through injuries received during or after birth, as through abrasions of the skin or by the entrance of septic matter derived from the discharges of the mother into the mouth, stomach, rectum, vagina or lungs of the infant. The water used for washing the infant may be the source of the infection. Operations performed on the new born, such, for instance, as circumcision, the opening of a cephalhematoma or the incision of the frenulum linguae for tongue-tie may serve as portals of entry. Although the possibility of the development of sepsis through such sources is to be recognized, in the great majority of instances it starts at the umbilicus, beginning oftenest before complete separation of the cord, and is first manifested by an umbilical arteritis. In 340 autopsies in new-born infants Runge² found 36 cases of general sepsis. In 30 of these infection could be traced to the navel, and in all of these umbilical arteritis was present. The fact that the umbilical wound is healed is no proof that infection of the umbilical vessels does not exist.

Beginning at the portal of entry, wherever this may be, the infection may either remain local, or may become more general, there occurring in the new born but little protecting lymphadenitis to prevent this; or general septic infection may occasionally take place without the portal of entry showing anything abnormal or even being discoverable. If the sepsis is local only, there may be produced phlegmonous inflammation of the injured skin of various parts of the body, mammitis, omphalitis, thrombosis of the umbilical arteries or vein, stomatitis, rhinitis, etc.; or in cases of aspiration of an infectious fluid, septic pneumonia. If the process extends from one of these portals of entry the sepsis may involve almost any of the more distant parts of the body.

In infants still-born or dying very shortly after birth, the subjects of intra-uterine infection, there is found macerated skin, petechiæ on the surface of the body and on the serous membranes, bloody or serous effusions into the serous cavities, and smaller or larger extravasations of blood

¹ Beiträge z. Geburtsh. u. Gynäk., 1906, X, 320.

² Krankh. d. ersten Lebenstagen, 1893, 136.

into and fatty degeneration of the internal organs. In those acquiring the disease after birth there is the same tendency to fatty degeneration of and hemorrhages into the internal organs, with petechiæ of the serous membranes, and bloody, serous, or purulent effusions into the serous cavities. There are also various lesions of different organs, depending upon the localization of the septic process. Septic thrombi are frequent, especially of the umbilical arteries. Inflammation or hemorrhage of the brain or meninges is also common, as are cutaneous or subcutaneous abscesses, peritonitis, pericarditis, pneumonia and pleurisy. Müller¹ regards peritonitis as one of the frequently observed localizations in general septic infection. Bednar² found croupous pneumonia 15 times and pleurisy 10 times in the autopsies on 87 cases of sepsis. Runge³ recorded pneumonia in 21 out of 55 cases of sepsis of umbilical origin. Involvement of the digestive apparatus is frequent. Osteomyelitis, arthritis, nephritis, pyelitis, and otitis are often observed.

The ultimate cause of the pyogenic infection appears to be chiefly varieties of the staphylococcus and streptococcus. The pneumococcus, colon bacillus, bacillus pyocyaneus, gonococcus and others are also factors in some instances. The germs, as stated, are acquired in many ways. The vaginal discharges are a fruitful starting point for the cases developed during birth. Later there is no dearth of sources from which infection can arise, all depending upon lack of aseptic cleanliness of some form. All new-born infants are prone to the development of the disease, but it is particularly common among those born in institutions, since the dangers of infection are greater there. It is disputed whether infection may take place through the milk of the mother.

Symptoms.—The symptoms usually appear either immediately after birth or at some time within the first 10 days. They vary greatly, depending upon the seat of the lesions. As a rule, however, in general septic infection there is great and characteristic depression of strength with rapid loss of weight and entire anorexia. Fever is irregular. It is generally present at the onset and frequently high, but may be absent throughout and the temperature is often subnormal toward the end. Severe diarrhea is a common symptom; vomiting may occur; icterus is frequent and often intense, or the skin may be of a pale-grey tint, and septic erythemata are frequently observed. The pulse is rapid and weak, the respiration irregular; sometimes deep and rapid. The occurrence of small or larger hemorrhages in different parts of the body is a common and characteristic symptom. The child looks ill, is usually apathetic or somnolent, has a feeble cry and sometimes develops the symptoms of collapse. Various nervous symptoms may be seen, among them tossing, rolling of the head, hypertonic states, twitching of the muscles, tremor, and sometimes convulsions. The urine generally contains albumin. Enlargement of the spleen may sometimes be discovered. The coagulation-time of the blood is increased.

When the disease is prolonged the symptoms may be masked, the principal one being a rapid, continuous loss of weight, until finally the development of some local septic process makes the condition clear. Naturally in many instances the character of the symptoms is modified by the development of those of localized involvement as well.

¹ Gerhardt's Handb. d. Kinderkr., 1877, II, 477.

² Krankheit. d. Neugeb. u. Säugling., 1850, IV, 245

³ *Loc. cit.* 95.

Course and Prognosis.—Some cases run a rapid course, ending fatally in from 1 to 3 days. Others last for weeks; but in all the prognosis is very unfavorable. The mildest and more slowly progressing cases may occasionally recover, but this is certainly unusual, and all those with well-marked symptoms of a general infection terminate fatality. The earlier the infection occurs and the more widespread the lesions, the worse the prognosis.

Diagnosis.—The diagnosis offers no difficulty where the symptoms are well developed, and a source of infection can be recognized. It is often impossible when no portal entry can be found which shows evidence of septic infection, or unless some local metastatic manifestation of sepsis develops. The discovery of enlargement of the spleen is corroborative but not positive evidence. The absence of fever does not exclude sepsis, and the failure of a leucocytosis to appear is not conclusive. The only positive evidence may be the discovery of septic germs in the blood.

Treatment.—This consists principally in prophylaxis. Every possible source of infection is to be removed. Should the mother be suffering from puerperal fever, the child must be separated absolutely from her. Any discoverable wound upon the skin or mucous membrane of the child must receive as careful antiseptic treatment as possible. The umbilicus (see p. 72) and the mouth should have especial attention. For the disease when once developed little can be done, except the employment of vigorous stimulating and supporting measures, the prevention of shrinking of the tissues by the employment of hypodermoclysis, and the adoption of such surgical treatment for local conditions as seems indicated.

To many of the special local manifestations of septic infections, such as omphalitis, inflammation of the umbilical vessels, mammitis, some forms of hemorrhage, and the like, separate consideration will be given among the diseases of the new born. Others, such as pneumonia, peritonitis, erysipelas, etc., will be discussed later in the general consideration of these disorders.

CHAPTER III

ACUTE FATTY DEGENERATION OF THE NEW BORN

(Buhl's Disease)

This condition was first described by Buhl in 1861.¹ Cases have been reported by a few others, notably Hecker² and Runge.³

Etiology.—The disease is a very uncommon one, or at least seldom recognized in human beings, although a similar condition in the new born of animals has been seen more frequently. The cause is entirely unknown. Although it is very probable that it is of a septic nature, this has not been proven, and has, indeed, been strongly contested. In a typical case described by Lucksch,⁴ colon bacilli were found in the blood. The disease occurs in isolated cases only, and has been seen especially in well-developed infants who have been born much asphyxiated without

¹ Klinik d. Geburtsk., 1861, I, 296. Ref. Runge, 162.

² Arch. f. Gynäk., 1876, X, 537.

³ Krankh. d. ersten Lebenst., 1893, 162.

⁴ Prag. med. Wochenschr., 1913, XXXVIII, 167.

discoverable cause, and have continued so to some extent in spite of treatment.

Pathological Anatomy.—The body is cyanotic and frequently icteric also. The skin is often edematous and may exhibit hemorrhages. Effusions of blood, either ecchymotic in character or in large amounts, are found in nearly all the internal organs; especially from or in the serous membranes of the brain, the pleura and pericardium, the endocardium, the mediastinal connective tissue and the peritoneum. They also occur in the muscles, the thymus gland, and most of the mucous membranes. The brain is soft and congested; the lungs often contain infarcts and the bronchi bloody mucus. The heart-muscle is firm and dark red in cases dying promptly; pale and soft in those of longer duration. The spleen is generally enlarged and very soft. The liver is at first dark red; later pale, icteric and perhaps slightly enlarged. The stomach and intestines often contain effused blood and there are hemorrhages in the mucous membrane, and the walls are thickened and edematous. The kidneys are swollen and dark-red in the early stages, but later pale and yellowish. They exhibit numerous hemorrhages into their substance, and often a complete choking of many of the tubules with granular fatty matter. The umbilicus and its vessels are normal, except for the evidences of the presence of hemorrhage which has occurred from it and in the tissues about it.

The change most characteristic of the disease is the marked fatty degeneration of the liver, heart, kidneys and epithelium of the pulmonary alveoli. This does not necessarily involve in each case all of the organs mentioned, but may in some of them be entirely absent or be replaced by a parenchymatous inflammation. The degenerative lesions are similar to those produced by phosphorus-poisoning.

Symptoms and Course.—The asphyxia with which these children are born does not yield to treatment, and many of the cases die promptly. If life is maintained longer there develops diarrhea often with bloody or blackish stools (melena), frequent vomiting of blood, and hemorrhage from the navel after the separation of the cord, this being generally in small amount, but sometimes large enough to be fatal. The hemorrhagic symptoms appear about the 5th day of life. The cyanotic color of the skin persists, becomes combined with or gives place to icterus, and edema is seen in some instances. Bleeding may occur into the skin or from the mucous membrane of the mouth or nose, conjunctiva or ear. There is anemia, great prostration, rapid loss of weight, and collapse. Fever is absent or inconsiderable. Not always are all the symptoms present. The asphyxia may be followed by sudden death, or may in some cases be slight at first and then may increase rapidly, accompanied by the characteristic symptoms. In some instances no external hemorrhage whatever is seen. The disease may last but a few hours before the fatal ending occurs. It rarely continues as long as 2 weeks. The **prognosis** is entirely unfavorable. Whether lighter forms recover cannot be known, since in these the determining diagnostic feature, the fatty degeneration, is necessarily undiscoverable.

Diagnosis.—An absolute diagnosis is impossible except by microscopic examination of the tissues. Even a probable diagnosis is difficult unless all the symptoms are present, especially the combination of marked asphyxia and widespread hemorrhage, and the absence of any umbilical disease. The disorder resembles closely and probably is identical with some forms of sepsis of the new born. Infectious hemoglobinemia

is like it in many particulars, but is distinguished by the characteristic condition of the urine present in this affection. In the case of infants which die asphyxiated soon after birth it is impossible to determine without autopsy whether acute fatty degeneration was not the disease present; and this applies, too, to those born little or not at all asphyxiated, who later suddenly develop this symptom and die promptly. The importance of the microscopic examination in all doubtful cases is, therefore, evident.

The **treatment** can be only symptomatic, efforts being directed to the sustaining of strength, the relief of the asphyxia, and the control of the hemorrhage.

CHAPTER IV

ACUTE INFECTIOUS HEMOGLOBINEMIA OF THE NEW BORN

(Winckel's Disease; Hemoglobinuria neonatorum; Cyanosis afebrilis icterica pernicioza cum hemoglobinuria; etc.)

Although Winckel¹ recognized the existence of hemoglobinuria as a symptom in an epidemic affecting 23 children in the Lying-In Hospital of Dresden, yet an earlier epidemic in which 10 children were attacked was well described by Bigelow in 1875.² A condition probably the same had already been reported by Parrot³ and by Charrin⁴ in 1873, and still earlier by Pollack⁵ in 1871.

Etiology.—The affection is a rare one. It occurs generally in institutions and in an epidemic form, although isolated cases have occasionally been reported, and it attacks well-developed children equally with others. It appears closely allied both to the septic infection and to the hemorrhagic disease of the new born, and is also very closely related to acute fatty degeneration of the new born (p. 260). That it is an infection seems certain, yet the nature of this is unknown; for although both streptococci and colon bacilli have been reported, these are germs commonly found in sepsis, which is without any such characteristic group of symptoms. That it is not produced by the ingestion of a poison was proven by Winckel's investigations.

Pathological Anatomy.—The lesions as based upon Winckel's description show the internal organs as well as the skin cyanotic and icteric in hue. The spleen is much enlarged and hard, of a blackish red color, and contains a large amount of brown coloring matter, partly free, partly in the cells of the pulp. The cortical layer of the kidney is thicker than normal, of a brownish color, and exhibits small hemorrhages. The pyramids are blackish-red and show narrow black streaks converging toward the papillæ and due to the deposit of hemoglobin in the canals. The bladder contains greenish-brown urine. The liver is enlarged; the mesenteric glands and Peyer's patches swollen. Punctate hemorrhages are seen in most of the organs of the body, as in the membranes of the brain and spinal cord, under the serous covering

¹ Deutsch. med. Wochenschr., 1879., V, 303.

² Bost. Med. and Surg. Journ., 1875, March 11, 277.

³ Arch. de phys. norm. et path., 1873, 512.

⁴ Thèse de Paris, 1873.

⁵ Wien. med. Presse, 1871, 457.

of the liver, and especially in the pleura, endocardium and pericardium and in the mucous membrane of the stomach and intestines. There is sometimes a fatty degeneration of the liver and heart-muscle. The umbilical vessels are nearly always normal. Bacteria of any sort have usually not been discovered.

Symptoms.—The disease begins usually on from the 4th to the 8th day of life, occasionally earlier or later. The earliest symptoms are restlessness, loss of appetite, prostration, and intense cyanotic discoloration of the entire surface. Well-developed icterus is promptly combined with this, and rapidly increases in intensity until the surface is of a bronze color. The temperature is normal or below, rarely slightly elevated. The respiration is generally accelerated, the pulse little if at all altered. The urine is pale-brown in color and voided frequently in small amounts with straining. Examination shows the presence of hemoglobin, granular casts and renal epithelium, urate of ammonium, micrococci, and a small amount of albumin. No bile or biliary acids are present in it. The stools vary in color from dark-green to yellowish or brown. Vomiting and diarrhea occur occasionally. On incising or scratching the skin over the most cyanotic regions a thick, syrupy fluid of a blackish-brown color exudes, but only on firm pressure. The blood shows an increase in the number of leukocytes, numerous granules, and a great diminution in the number and increase in the size of the red blood-cells, many of which appear to have lost their coloring matter. Collapse develops with great rapidity; there is somnolence, and convulsions are liable to terminate the case.

Prognosis.—This is most unfavorable, as all severe cases die. Only 2 of Bigelow's and 2 of Pollock's cases recovered; while 19 of Winckel's 23 cases are known to have ended fatally, and only 1 certainly to have recovered. Ljwow¹ reported better results, 4 out of 7 cases recovering. Death usually occurs in a few hours to 4 days, generally in 2 days.

Diagnosis.—The disease is so characteristic that mistakes in diagnosis can hardly be made. Although it resembles strongly in some respects acute fatty degeneration of the new born, it is to be distinguished by the presence of hemoglobinuria and the occurrence usually in epidemics.

The **treatment** can be only symptomatic, efforts being made to maintain life by stimulants and food.

CHAPTER V

HEMORRHAGE IN THE NEW BORN

Hemorrhages at this early period of life, although not common, are still frequent as compared with their presence later in childhood. They may occur in different parts of the body and from different causes. Those not mentioned here will be found in other chapters.

Etiology.—The previous general health appears to have no constant influence, for while some of the infants affected have previously been in poor condition others appear strong and hearty. In some instances the hemorrhages, usually single, seem to be the result of accident, as in hematoma of the scalp (p. 269) or of the sternocleidomastoid (p. 272), and in some cases of hemorrhage from the umbilicus (p. 292) or from some

¹ Medicinskoje Obosrenje, 1893, No. 14. Ref. Jahrb. f. Kinderh., 1894, XXXVIII, 497.

other part of the body as a result of trauma received. After difficult and prolonged labor various visceral hemorrhages may occur, especially those within the cranium. Spencer¹ concludes from 150 autopsies that the use of the forceps is a common producer of intracranial hemorrhage. Premature birth is another cause of hemorrhage, especially within the cranium. Difficult breech presentations are more prone to occasion hemorrhages of the abdominal viscera. In very many instances the bleeding depends upon septic infection, as already pointed out, and bacteria of various sorts have been found in the blood. (See p. 259.) In these cases the hemorrhages are liable to be widespread and of small size. In others special hemorrhage-producing bacteria have been found (Schloss and Commiskey).² Congenital syphilis appears to be the active agent in some instances. The influence of this factor has been reviewed by Hess³ and by Pontoppidan.⁴ Wilson⁵ reported hemorrhage occurring in 45 of 3364 new-born infants, and in 10 of these it appeared to be due to congenital syphilis. In many others we are ignorant of the exact etiology and pathology, although the presence of some infection or toxemia seems probable. Either the resistance of the blood-vessel walls is weakened in some way, or the blood itself is altered. Studies made by Schloss and Commiskey⁶ upon the coagulability of the blood in 10 cases of hemorrhagic disease found this diminished in some instances, but unaltered in others. Many of the forms of hemorrhage will be referred to later in discussing the diseases of the organs in which the bleeding occurs.

Bleeding which takes place without any evidence of trauma may be called "spontaneous." Townsend⁷ has applied the term "**The hemorrhagic disease of the new born**" to the condition in which small or large effusions of blood occur simultaneously in many different parts of the body, independent of any discoverable cause. It was earlier described by Minot in 1852.⁸ The affection appears to be probably of an infectious nature and self-limited, most of the non-fatal cases recovering within a week. This early spontaneous hemorrhage is quite distinct from hemophilia (Vol. II, p. 474), among other respects in that the tendency of the new born to bleed does not persist if the child survive. Hemorrhage in the new born occurs much most frequently in institutions. Ritter⁹ found 190 such in 13,000 infants in the Prague Foundling Hospital; *i.e.* 1.46 per cent., and Townsend¹⁰ 32 cases in 7225 births; *i.e.* 0.44 per cent. The accident occurs much the most frequently in the 1st or the 2d week of life. Abt¹¹ estimates that from 1 in 500 to 1 in 700 institution-infants suffer from hemorrhage.

Locality of the Lesions.—The regions of the body affected are various and generally are multiple. One of the commonest seats is the gastroenteric tract. In Ritter's¹² 190 cases hemorrhage occurred from the intestine in 39, the mouth in 28, and the stomach in 20. (See Melena,

¹ Trans. Obst. Soc., Lond., 1891, XXXIII.

² Amer. Jour. Dis. Child., 1911, I, 276.

³ Archives of Pediatrics, 1904, XXI, 598.

⁴ Hospitalstidende, 1916, LIX, 626. Ref. Brit. Jour. Child. Dis., 1916, XIII, 308.

⁵ Archives of Pediatrics, 1905, XXII, 43.

⁶ Amer. Jour. Dis. Child., 1912, III, 216.

⁷ Boston Med. and Surg. Jour., 1891, CXXV, 218; Archives of Pediatrics, 1894, XI, 559.

⁸ Amer. Jour. Med. Sci., 1852, XXIV, 310.

⁹ Oesterreich. Jahrb. f. Pädiatrik, 1871, I, 127.

¹⁰ Loc. cit., 218.

¹¹ Jour. Amer. Med. Assoc., 1903, XL, 284.

¹² Loc. cit., 159.

p. 266.) The flow of blood is generally small, but is often in larger amount and discharged from the bowels as a black, tarry substance. Bleeding may take place into other of the abdominal organs, due generally to difficult labor when the hemorrhage is single, but oftener the result of the hemorrhagic disease, sepsis, or other causes when the hemorrhage is multiple. Large effusion into the suprarenal capsule is one of the most frequent forms (Vol. II, p. 529). Small hemorrhages under the peritoneum are seen not infrequently. Larger ones also may occur here, being commonly traumatic in origin. These and especially those into the suprarenal bodies may burst into the peritoneal cavity and produce sudden death from collapse. In some such instances it may be impossible to discover the original source. Occasionally bleeding may take place from the liver (Bonnaire and Durante).¹ Intracranial hemorrhage is one of the commoner varieties. In 33 autopsies upon infants born prematurely, Couvelaire² found the brain to be the seat of hemorrhage in 5 instances, excluding cases of meningeal and intraventricular bleeding. The numerous instances of cerebral birth-palsy are generally dependent upon intracranial bleeding. (See Cerebral Palsy, Vol. II, p. 365.)

Small hemorrhages may occur upon the serous covering of the lungs. More rarely larger ones take place into the pulmonary tissue, the bronchi, or the pleural cavity. Epistaxis is uncommon. Small effusions of blood may be found in the thymus gland. Hemorrhage, generally in small amount, may occasionally arise from the female genitals. Schukowski³ collected only 35 cases of metrorrhagia in 10,000 new-born female infants. (See also Vol. II, p. 230.) Bleeding from the bladder or kidneys may exceptionally occur. This may be the result of a general hemorrhagic disease or may be purely traumatic in origin, dependent upon the presence of uric acid infarcts. Hemorrhage may take place from the ears or the eyelids.

Bleeding from the umbilicus is the most common variety, 132 (69.47 per cent.) of Ritter's⁴ 190 cases being of this nature, and in 97 the hemorrhage being limited to this region. It may occur as a result of accident or as a manifestation of a general hemorrhagic condition (see Umbilical Hemorrhage, p. 292) and is very often combined with bleeding from other parts of the body. Widespread subcutaneous hemorrhages are not uncommon. They develop oftenest in parts most pressed upon, although other regions are not spared. Bleeding from the skin following a slight wound, as for an examination of the blood, is not infrequent in hemorrhagic cases.

Symptoms.—The *traumatic* hemorrhages, due as they generally are to injury at birth, as a rule reveal themselves promptly, except those of the abdominal and thoracic viscera, where no symptoms at all may appear until, perhaps, a sudden collapse occurs followed promptly by death. Those of a *spontaneous* nature manifest themselves generally by visible bleeding in several regions of the body. Inasmuch as infants tolerate loss of blood very badly, a comparatively small hemorrhage may readily produce weakness of pulse, great depression of strength and loss of weight. Anemia is a natural result. The temperature may be elevated, but is often subnormal. Icterus is a common symptom. It was seen in 21 per cent. of Ritter's cases. As a rule the amount of blood lost at one time is small. It is the repeated losses, and especially losses in many parts of the body, which occasion the general symptoms in most cases.

¹ L'Obstetrique, 1911, IV, 825.

² Ann. gynéc. et d'obstet., 1903, LIX, 253.

³ Sparda Vop., 1902, II, 3., Ref. Jahrb. f. Kinderh., 1903, LVII, 105.

⁴ Loc. cit., 190.

Prognosis and Course.—These depend much on the cause, on the amount of blood lost, and on the duration of the process. The mortality was formerly high; 75.79 per cent. in Ritter's cases of spontaneous hemorrhage and 62 per cent. in Townsend's 50 cases; while in 609 published reports of hemorrhage collected by the latter writer the mortality was 79 per cent. Of recent years improved methods of treatment have decidedly diminished the death-rate. Death in the fatal cases generally occurs within a week. It may be very sudden from collapse when the hemorrhage is large.

Diagnosis.—Where the bleeding is evident to the eye the diagnosis is easy. Where it is concealed it is difficult and often impossible. The determining of the nature of the cause is important. As a general rule large single hemorrhages are the result of local processes, while widespread multiple hemorrhages depend upon sepsis, syphilis, or the general hemorrhagic condition referred to. Of course, the discovery of blood effused in or from only one part of the body does not prove that concealed visceral bleeding may not be taking place as well.

Treatment.—Local measures to arrest bleeding are indicated where applicable. Among these are the use of astringents, especially the application of a caustic, such as nitrate of silver in solid form, liquor ferri subsulphatis or chromic acid. The exhibition of gelatine in 10% solution administered internally freely has been largely employed, and appears to be of value. From 10 to 50 c.c. (0.34 to 1.69 fl.oz.) may be given subcutaneously once or twice a day with the greatest caution to obtain complete sterilization and to follow aseptic precautions, lest septic infection or even tetanus result. Internally gelatine may be administered either by the mouth or by the rectum. Adrenalin chloride has been recommended, giving 1 to 4 minims (0.062 to 0.246) of a 1:1000 solution internally. Calcium chloride or lactate in amounts of 20 to 40 grains (1.3 to 2.6) in 24 hours in divided doses may also be employed. One of the best of remedies is the administration of a blood-serum. Human blood from a parent may be given by direct transfusion, or this or the serum be injected subcutaneously, as recommended by J. E. Welch¹ in quantities of from 10 to 30 c.c. (0.34 to 1.01 fl.oz.) every 4 to 8 hours. To prevent clotting the blood before injection may be mixed with a 2 per cent. sterilized sodium-citrate solution in the proportions of 1:10. (See p. 246.) Should human blood not be available, horse-serum or rabbit-serum may be given subcutaneously in the same amount, diphtheria-antitoxin being selected for this purpose if nothing else can be obtained. In the way of general treatment the loss of blood must be made up by subcutaneous or rectal injections of normal salt-solution, the temperature maintained, and the strength supported.

MELENA NEONATORUM

In this form of hemorrhage in the new born are grouped cases which are quite dissimilar in origin and nature, but which show the characteristic symptom, *i.e.*, the discharge of black altered blood (*μέλαινα*: black) from the intestines or, by vomiting, from the stomach. The condition was first described by Ebart in 1723.² The title should be reserved to describe the symptom merely; not the cause.

¹ Amer. Jour. Med. Sci., 1910, CXXXIX, 800.

² Wiederhhofer, Gerhardt's Handb. d. Kinderkr., IV, 2, 408.

Etiology.—The affection is sometimes divided into *Melena spuria* and *Melena vera*. In the former the blood enters the infant's stomach or intestines from outside sources, as from a hemorrhage from the mouth, nose or lungs, a wound in the nipple of the nurse, or from the swallowing of maternal blood during labor. In *melena vera*, which is the form about to be considered, it comes from the effusion of blood into the stomach or intestines. Even the cases of the latter group may be divided into the *symptomatic*, in which other manifestations, often hemorrhagic, of a constitutional disease are present, and the *idiopathic*, in which the melena is the only symptom of a hemorrhagic condition observed.

Melena is a comparatively uncommon disorder occurring about once in every 1000 or 2000 births, according to different estimations (Gerhardt-Seiffert).¹ The etiology is not clearly understood, and certainly varies with the case. Lesions of the mucous membrane or of the deeper vessels of the gastroenteric canal are prominent causes, but the method of production of these lesions is far from clear. Often they are the result of congestion, which may itself be brought about in various ways incident to birth, such as prolonged asphyxia, violent efforts at extracting the child or at its resuscitation, compression of the umbilical cord, congenital diseases of the heart, congenital syphilitic hepatitis, and the like. In other instances some constitutional condition has produced a weakness of the vessels or an alteration of the blood itself; among these causes being syphilis and sepsis, the melena being then only one of the evidences of the hemorrhagic tendency present. A delay in the coagulation of the blood is said by Lövegren² to be present. Various bacteria have been reported as found from time to time but their relationship to melena has never been satisfactorily proven. It is probably very close in some instances, but of no bearing in others. The influence of the development of ulcers in the gastroenteric tract, especially the duodenum, in giving rise to gastrointestinal hemorrhage is easy to appreciate, but the method of production of such ulcers is not well understood; whether embolic, or dependent on erosion after hemorrhage into the mucous membrane. (See Ulcer of the Duodenum, p. 797.) Ulceration is, however, not a common post-mortem finding in fatal cases of melena. In many instances it would appear as though the blood makes its way from the vessels through the mucous membrane of the intestine without any discoverable lesion.

Pathological Anatomy.—The surface of the body and the internal organs are anemic, and the gastrointestinal canal is found filled with blackish fluid. Beyond these there is no uniformity in the conditions present. Occasionally ulcers are discovered on the mucous membrane of the stomach or intestine, especially the duodenum. They may be very minute or larger, and sometimes exhibit the characteristic appearance of the ordinary peptic ulcer. In some cases only minute erosions and in others only minute extravasations of blood are found. Vorpahl³ reported an instance of melena dependent upon rupture of dilated veins in the esophagus. In the large majority of cases nothing whatever of moment is discovered in the gastrointestinal tract at autopsy. Careful study of other parts of the body may reveal the evidences of sepsis or of syphilis, or the characteristic lesions of acute fatty degeneration.

Symptoms.—Although in a certain proportion of cases there is some asphyxia at birth, in the large majority the child is of healthy

¹ Lehrb. d. Kinderkr., 1897, 71.

² Jahrb. f. Kinderh., 1914, LXXIX, 708.

³ Arch. f. Gynäk., 1912, XCVI, 377.

appearance. The discharge of blood begins usually on the 2d day of life and seldom later than the 4th day. In most cases the blackish or blackish-red matter is both vomited and evacuated by the bowel, but in many instances it is passed from the bowel only. In an analysis by Vassmer¹ of 67 published cases blood was found in the stools alone in 20, in the vomitus alone in 6, and in both in 37. The amount lost may be slight, but it is oftener so large that the infant rapidly becomes very anemic, apathetic, prostrated, with feeble cry, and finally collapsed. There may be slight transitory fever, or normal or subnormal temperature. As a rule there is no distention or tenderness of the abdomen. The disease seldom lasts longer than 2 to 3 days. In the cases which survive recovery is rapid, except from the anemia, unless some constitutional disorder, such as syphilis, is accountable for the hemorrhage and keeps the child ill in other ways. The disappearance of the anemia, of course, requires a longer time.

Prognosis.—The death-rate is high, probably between 50 per cent. and 60 per cent. (56 per cent. Silbermann).² In the cases of idiopathic melaena vera; *i.e.* those in which the hemorrhage is limited to the gastrointestinal tract and is not a symptom of a general hemorrhagic diathesis, the mortality is distinctly less than the figures given. The mortality is less also, in the cases where the hemorrhage is from the bowel only than when there is hematemesis. Thus in the 20 cases of Vassmer's series in which the hemorrhage was from the bowel alone the mortality was 10 per cent. The liability of death depends largely on the cause of the bleeding and on the amount of it.

Diagnosis.—Melaena vera is to be sharply distinguished from the spurious melaena referred to. In the latter the hemorrhage is seldom large; it does not always begin so early in life; and the child does not, as a rule, appear ill. Examination of the nipple of the mother and of the nose and mouth of the child may discover the source. When melaena is limited to discharge of blood from the intestines it may be readily confounded with the passage of meconium, especially in mild cases, and be easily overlooked. The material is, however, evacuated more frequently in cases of melaena, and the slightly reddish color of the blackish masses or of the diaper serves to distinguish it. The use of the microscope, especially the combined chemical and microscopical examination for blood corpuscles and hemin crystals, makes the diagnosis positive. Finally, the presence of hemorrhages from other parts of the body or of symptoms other than those described will remove the case at least from the category of idiopathic melaena vera. Occasionally the gastrointestinal hemorrhage is concealed, and the child dies without any external bleeding having taken place. In such instances an antemortem diagnosis is not possible.

Treatment.—This consists in supporting the strength and checking the hemorrhage. Stimulants, digitalis, the maintaining the temperature, and the administration of food are required. Cold to the abdomen may check the hemorrhage, but is generally poorly tolerated by infants. Tincture of the chloride of iron (1 minim) (0.062) or fluidextract of ergot (2 to 5 minims) (0.123 to 0.308) has been recommended. Calcium lactate, 20 to 40 grains (1.3 to 2.6) in 24 hours, appears serviceable. Gelatine seems to be the best of remedies. It may be given by the mouth in a 5 to 10 per cent. solution, as for any form of hemorrhage in the new

¹ Arch. f. Gynäk., 1909, LXXXIX, 275.

² Jahrb. f. Kinderh., 1877, XI, 378.

born. (See p. 266.) The subcutaneous injection of human blood or serum, or of horse or rabbit serum, has likewise been proven valuable (p. 266) in the hands of many physicians.

CEPHALHEMATOMA

By this title is designated a form of hemorrhage in the new born producing a fluctuating swelling situated between the bones of the skull and the overlying tissues (cephalhematoma externum). Sometimes the blood is effused within the skull between the bone and the dura mater (cephalhematoma internum). The description which follows applies almost entirely to the external variety.

Etiology.—The disease is not very common. Hennig¹ found it recorded 230 times in 53,506 newborn infants, *i.e.* 0.43 per cent., and Hofmohl² 371 times in 59,885 cases; *i.e.* 0.6 per cent. Almost always the condition develops in the first few days of life, but very rarely cases have been recorded occurring in the first weeks or months, or even later in infancy. (Friedmann³—case in a child of 4 years.) Males are much oftener affected.

The direct cause of cephalhematoma is often some injury received during birth. Thus it occurs much most frequently in the children of primiparæ (34 out of 40 cases, Meyer⁴), and in vertex presentations when pressure has been long continued. The circular pressure exercised by the uterus and the absence of this over the central presenting part, probably tends to make the vessels yield and break. Yet in very many instances no evidence of direct trauma can be discovered, and the lesion appears to be due entirely to other causes. The condition may even develop in premature infants or after very easy labor, or sometimes in breech presentations. The presence of asphyxia tends decidedly to produce it, and it may also be one of the evidences of a general hemorrhagic disorder in the new born. The looseness with which the periosteum is attached to the underlying bone in the new born, and the delicacy of the blood-vessel walls, also predispose to its development.

Pathological Anatomy.—The lesion is generally unilateral, yet it may be bilateral, and rarely even appears over 3 different bones. The seat of the swelling is much more frequently over the right parietal bone. Hennig⁵ reported it here in 57, and over the left parietal bone in 37, out of 127 cases. The hemorrhage takes place below the periosteum, lifting this from the bone possibly as far as the sutures, where it adheres too firmly to permit of it. The blood remains fluid sometimes even for some weeks. It averages from 0.5 to 1.5 fl.oz. (15 to 44) in amount, but it may be much more than this. In recent cases it is discharged with a spurt when incision is made. The bone is pale, rough, and often covered with fibrinous clots. An exudate of new osseous tissue forms a peripheral boundary-ridge upon the bone about the tumor. This is soft and velvety in recent cases, but later hard and projecting distinctly above the surface of the skull.

The periosteum over the swelling is bluish and often covered with small effusions of blood. In advanced cases small plates of new bone

¹ Gerhardt's Handb. d. Kinderkr., II, 49.

² Arch. f. Kinderh., 1880, I, 309.

³ Münch. med. Wochenschr., 1904, LI, 387.

⁴ Hospitalstidende, 1897, IV, 585.

⁵ *Loc. cit.*

are deposited here and there in the periosteum. Section of the scalp over the swelling shows numerous scattered punctiform hemorrhages.

Quite exceptionally the bleeding instead of taking place under the periosteum occurs under the aponeurosis (*cephalhematoma subaponeurotica*). In such cases, of course, no bony ridge exists and the tumor is not necessarily limited by the sutures. Both varieties may occur together. Infrequently examination within the skull reveals also a hemorrhage between the dura mater and the bone, corresponding in position to the hematoma outside and perhaps connecting with it. McKee¹ found this internal cephalhematoma reported in but 16 instances in medical literature, in 11 of these associated with the external variety. When there has been decided trauma during labor fissures or fractures of the bones of the skull may be present. In the cases depending upon constitutional conditions hemorrhages occur in other parts of the body as well.

Symptoms and Course.—Although generally discovered 2 to 3 days after birth the condition begins earlier than this but is not noticed.



FIG. 46.—CEPHALHEMATOMA.
(From a photograph.)

Sometimes it is at first concealed by a caput succedaneum, and only becomes visible as this disappears. It is at first small, but grows rapidly. Its maximum size is attained in from 6 to 8 days, the bleeding then ceasing, and the tumor varying in size up to that of a large hen's egg (Fig. 46). It is at first rather flat and soft, but soon becomes rounded and tense although fluctuating. It is not hot or tender to the touch and rarely pulsates. It cannot be reduced by pressure, and is not affected by crying. The skin over it is sometimes darker

in color than normal but otherwise unaltered in appearance. The change in color is observed especially when the hemorrhage is subaponeurotic. The tumor covers all or a part of one of the bones of the skull, but if subperiosteal never passes beyond the sutures or over the fontanelle. Where the swelling appears to cover the adjoining bone also it is really a double cephalhematoma, and a distinct groove can be felt beneath the two at the position of the suture.

After the tumor has existed from 2 to 3 days, a soft ridge can be felt forming around its periphery. This later becomes harder and thicker as osseous tissue develops in it, and gives very much the sensation of a soft mass protruding through a large hole in the skull. The tumor maintains its size unabated until its 2d week and then begins slowly to grow smaller and less tense. As this occurs the bony periphery broadens toward the centre. A parchment-like sense of crepitation can now often be developed on palpation, dependent upon the new osseous growth in the periosteum.

Although recovery is exceptionally more rapid, as a rule absorption of the effused blood goes on very slowly and the tumor does not disappear for 2 or 3 months from the onset. The bony ridge may be still felt more

¹ Cincin. Lancet-Clinic, 1883, XI, 317.

or less distinctly for months more, or the whole region of the tumor may be left somewhat thickened. General symptoms are entirely absent.

Occasionally the course is not so favorable. Suppuration may take place within the tumor, especially if there are external wounds of the scalp present. In this event the swelling becomes red and tender, and the infant is evidently ill with the general symptoms of suppuration. The abscess which forms may discharge externally and be followed by healing, or it may give rise to a diffuse inflammation of the tissues of the scalp with involvement of the bone and secondary meningitis. In other cases the prolonged suppuration produces fatal exhaustion or general sepsis.

Sometimes the disease is complicated by cerebral symptoms from the beginning. In such instances it is probable that there is an internal cephalhematoma or a meningeal hemorrhage also present.

Prognosis.—This is entirely favorable in every uncomplicated case, and where the hematoma is not the result of a general disorder. The development of suppuration in the tumor is a serious complication, but a rare one, and makes the prognosis doubtful. Still more serious is the presence of a complicating hematoma within the cranium. These cases are fortunately exceptional, since they nearly always die.

Diagnosis.—Between the subperiosteal and the subaponeurotic cephalhematomata the distinction may be made by the presence in the former of a distinct, hard, marginal ring, the limitation by the sutures, and the absence of decided discoloration of the skin. Hematoma may be readily confounded with *caput succedaneum* during the first few days of life. This condition consists of an edematous swelling of the connective tissue of that portion of the scalp which has been presenting through the patulous os uteri, and is the result of the obstruction to the circulation at the periphery and the absence of pressure over the swollen region. The fact that it disappears in a few days, and the absence of fluctuation and of the development of any marginal wall also serve to distinguish it from cephalhematoma. *Meningocele* and *encephalocele* resemble cephalhematoma to some extent. They may exhibit pulsation, are influenced by respiration and by crying, are to some extent reducible often with the production of convulsions, and correspond in position with a suture or a fontanelle. (See *Meningocele*, Vol. II, p. 311.)

Abscess of the scalp is attended by heat, tenderness, and discoloration of the skin, with constitutional symptoms. A telangioma resembles a hematoma but slightly. There is no bony wall or fluctuation, while crying makes it larger and pressure somewhat smaller and the skin is discolored.

Treatment.—The best treatment, as a rule, appears to be a wholly expectant one. The child should be so placed that no injury to the hematoma can occur, and recovery entrusted to time. No applications are required. Many writers have urged early incision in order to drain away the fluid blood and hasten recovery. This, however, adds an unnecessary element of danger, in that there is the possibility of producing sepsis through the open wound, beside the chance of having a secondary hemorrhage occur. The expectant treatment although slow is safe. On the slightest evidence of suppuration beginning the hematoma should be incised and drained antiseptically. If there are indications of intracranial pressure, and an internal cephalhematoma communicating with an external one is suspected, the question of aspiration by puncture with antiseptic precautions is to be considered.

HEMATOMA OF THE STERNOCLEIDOMASTOID MUSCLE

This consists of a hemorrhage into the body of the sternocleidomastoid muscle. It is of rare occurrence and is the result of injury received during birth. In about 75 per cent. of the cases this is produced by traction upon the legs in breech presentations, as a result of which the blood-vessels in the neck are torn and a blood-tumor results. It may be brought about also by the pressure of forceps in vertex presentations or may occur without any cause for the lesion being discoverable.

The tumor produced is quite small, not over $1\frac{1}{2}$ inches (3.8 cm.) in length. It is tender on pressure due to the attending myositis, and at first soft. The skin over it is not discolored. Soon it becomes hard and the tenderness disappears as cicatricial changes take place in it. It may occupy any position of the muscle but is oftenest in the middle or upper part, and is most frequently situated upon the right side, only exceptionally occurring upon both sides. In 38 cases reported by Henoch¹ the right side was involved in 31. Although produced at birth it generally does not become apparent until the age of 2 weeks or later, especially if the neck is fat. The rigid inclination of the head of the infant toward the affected side with the chin turned toward the other is a symptom often present which leads to the examination of the neck and the discovery of the swelling.

The immediate **prognosis** is nearly always good. The swelling slowly grows smaller, although it may not entirely disappear for a few months. It is probable, however, that the rigidity of the neck may in rare instances remain throughout life. The cases of so-called congenital torticollis are probably produced in this way. (See Torticollis, Vol. II, p. 411.)

No **treatment** except rest is required in the acute stage. Later gentle massage and passive movements of the head may be of benefit. Should persistent torticollis remain surgical treatment may be demanded later, in the form of orthopedic apparatus or of operative interference.

CHAPTER VI

ICTERUS IN THE NEW BORN

Icterus is of very frequent occurrence in the new born, and may depend upon various causes. It may conveniently be divided into (1) symptomatic, and (2) idiopathic icterus.

1. *Symptomatic icterus* depends upon such general diseases as infectious hemoglobinemia, acute fatty degeneration of the new born, sepsis, syphilitic hepatitis, congenital cirrhosis of the liver and congenital obliteration of the bile-ducts. These are all of rare occurrence. The ordinary gastroduodenal catarrh with closure of the common bile-duct, which occasions icterus so frequently in adult life, is often a cause in the new born also and is liable to be more severe than in later life. Most of the forms of symptomatic icterus are discussed elsewhere in course, with the exception of that depending upon the congenital obliteration of the ducts to which brief special consideration may be given.

¹ Vorlesungen über Kinderkrankheiten, 1895, 35.

In symptomatic icterus the discoloration of the skin is generally very decided, and there are other symptoms of the causative disease evident. The urine often shows the presence of bile, and, in obstructive jaundice, the feces often the absence of it.

2. In *idiopathic icterus* in the new born there is present no discoverable congenital or acquired anatomical defect which can produce jaundice by obstruction, and no constitutional disease to account for it. It appears to be almost of a physiological nature, and there are no other symptoms attending it. To this form the title "*Icterus Neonatorum*" is commonly applied.

CONGENITAL OBLITERATION OF THE BILE-DUCTS

(Congenital Biliary Cirrhosis)

This is a rare condition of which, however, according to Thomson¹ more than 100 cases had been reported at the date of his writing. Holmes² in a very careful review of the subject published in 1916 estimates that nearly 120 cases have been reported. I have observed 2 instances of it, verified by autopsy.³ Congenital biliary cirrhosis, although described as a distinct affection, would seem probably, as claimed by Skormin,⁴ and by Rolleston and Hayne⁵ to be of the same nature as obliteration of the bile-ducts, both in pathogenesis and in symptomatology.

Etiology and Pathology.—The cause is little understood. The disease is liable to occur in several members of one family, this supporting the view that there exists some congenital failure in development. Some cases indicate the existence of a prenatal obliterative inflammation of the bile-ducts, which either starts as a biliary cirrhosis and descends to the larger ducts, or in which the two regions are affected at the same time. Still another view makes the cirrhosis secondary to complete obliteration of the main ducts and dependent upon the interference with the exit of bile. One of my cases supports this view. Thomson has shown that a biliary cirrhosis was present in nearly all of the reported cases which he collected.

It does not seem likely that syphilis plays any important part in the etiology. Some of the instances of severe congenital icterus may possibly be due to a partial obliteration of the bile-ducts involving the mucous membrane of the ducts and to a certain extent the connective tissue as well, but never advancing to complete obstruction. This theory would explain certain cases of jaundice reported by Arkwright,⁶ Weber⁷ and Pearson.⁸ It is certain that the obstruction may occasionally not be complete at first, and that entire obliteration may develop only considerably later.

Pathological Anatomy.—The bile-ducts may be undiscoverable or they may be replaced by a fibrous cord; or there may be obliteration at but a single point, the ducts above this being without stenosis. The

¹ Allbutt and Rolleston, *System of Med.*, 1908, IV, 1, 103.

² *Amer. Jour. Dis. Child.*, 1916, XI, 405.

³ *Arch. of Ped.*, 1905, XXII, 257. *Ibid.*, 1908, XXV, March.

⁴ *Jahrb. f. Kinderh.*, 1902, LV1, 203.

⁵ *Brit. Med. Journ.*, 1901, I, 758.

⁶ *Edinburgh Med. Journ.*, 1902, LIV, 156.

⁷ *Edinburgh Med. Journ.*, 1903, LV1, 111.

⁸ Underwood's *Dis. of Child.*, 1846, 10th Edit., 168.

gall-bladder may be absent or distended. The liver is generally enlarged, firm, and of normal or greenish color, and in most cases exhibits the histological lesions of hypertrophic cirrhosis. The blood-vessels are seldom involved; the spleen is enlarged.

Symptoms.—The principal symptom is icterus, which is present at birth or develops within the 1st week or occasionally later. The color is intense and often of a greenish hue. The occurrence of acholic stools is also of importance. Sometimes ordinary yellow movements are present at first, to be replaced later by the characteristic appearance. The passage of green feces does not, of course, necessarily indicate the presence of bile. The urine is intensely bile-stained; fever is not a symptom; but vomiting is liable to occur. Hemorrhages from the umbilical cord and into different parts of the body are a very characteristic symptom, yet in no way pathognomonic, as they may appear in severe icterus from other causes. Emaciation develops if the case is at all prolonged.

Course and Prognosis.—The outcome is necessarily fatal, if we include among the cases only those in which complete organic obliteration finally occurs. The course is seldom acute. Death takes place in convulsions or stupor, generally only after several weeks or even months, although sometimes the child dies in the 1st week of the disease.

Treatment can be only palliative and symptomatic. Operative interference at this early age appears to offer little hope; both on account of the difficulties attending it, and because biliary cirrhosis is usually combined with the obliteration.

ICTERUS NEONATORUM

Etiology and Pathogenesis.—The condition is a very common one, ranging according to different statistics from 15.69 per cent. (Seux)¹ to 84.46 per cent. (Cruse)² of all births. It is probable that careful observation would show it present to at least a slight degree in the great majority of the new born. Boys are oftener affected than girls; the first-born children oftener than later ones, and premature, light-weight, or atelektatic infants oftener than others (Kehrer).³

Regarding the exciting causes of the condition and the method of its production the years of investigation have not yet given an entirely satisfactory solution. The different theories group themselves for the most part into two classes: first, that the icterus is hepatogeneous in origin; *i.e.* arising from some disturbance in the secretion of bile in, or its exit from, the liver; second, that it is hematogeneous; *i.e.* depending upon degeneration of the blood-corpuscles in the circulation and the production in this way of biliary coloring matter. Although the jaundice may perhaps be in part of hematogeneous origin, it is certainly not entirely or even chiefly so, since in icterus neonatorum biliary acids are found in the serous fluids of the body (Runge)⁴ and it is admitted that these must have their origin in the liver. The method, however, in which the hepatogeneous icterus is produced in the new born is uncertain and various theories exist. Silbermann⁵ maintains that there is an abundant breaking up of red blood-cells in the circulation after birth. This produces an

¹ Recherches sur les mal. des enfants nouveau-nés, 1855, 269.

² Arch. f. Kinderh., 1880, I, 353.

³ Oesterreich. Jahrb. f. Pädiat., 1871, II, 71.

⁴ Krankh. der ersten Lebenst., 1893, 225.

⁵ Jahrb. f. Kinderh., 1887, XXVI, 252.

increase of material from which bile is constructed, and, in addition, an increase of fibrin ferment, which, on its part, occasions great congestion in the blood-vessels of the liver, and a consequent compression of the biliary capillaries. Yllpö¹ and Hirsch² have shown that the blood-serum of the fetus in its last month contains much more biliary pigment than is present in that of adults, and that the amount increases still more in the first days after birth. The liver is functionally immature, and is therefore unable to dispose of the excess of pigment, if this is unusually great, which consequently enters the blood-current instead of the biliary capillaries, and produces jaundice. When the amount of pigment exceeds 125 milligrams per 100 c.c. of blood the infant becomes icteric (Yllpö). The functional inability of the liver to dispose of the very large secretion of bile in the new born is confirmed by the studies of Hess.³

Among other opinions of the etiology, Birch-Hirschfeld⁴ believes that icterus is due to a compression of the larger biliary ducts by an edema of Glisson's capsule, the edema being dependent upon venous congestion incident to the circulatory changes following birth. Frerichs⁵ and others, on the other hand, consider the icterus caused by a diminution of venous blood present in the liver, the result of the cutting off of the blood-supply which had been received through the umbilical vein. A resorption of bile follows this. Still another view which has many adherents is that of Quincke⁶ that the icterus depends upon an increased production of bile derived from the meconium, the resorption of this from the intestine, and the direct passage of it through the patulous ductus venosus into the general circulation.

No theory advanced has not met with opponents, and the question still remains an open one to a large extent.

Pathological Anatomy.—Inasmuch as the condition is not a fatal one, autopsies can be made only on those infants who have died from other causes. The skin, subcutaneous connective tissue, lining of the arteries, and most of the serous membranes and exudates are yellow; the extent of this varying with the case. The brain, spinal cord, spleen, kidneys and liver are only slightly discolored if at all. The ductus choledochus is patulous; the kidneys are congested and exhibit uric acid infarcts and sometimes infarcts of biliary coloring matter, and the latter have also been found in the brain.

Symptoms.—The discoloration begins oftenest on the 2d or 3d day of life; much less often earlier or later than this. The surface of the body is the region most markedly affected, but in most cases the scleræ are also involved. The mucous membranes also are icteric. The discoloration is first noticed on the face or chest, and its degree varies greatly. Sometimes it is intense, but often it is very slight and can only be appreciated in good daylight and after the red color of the skin is removed temporarily by pressure with the finger. The urine is generally free from bile-pigment, at least by ordinary tests, although the microscope may sometimes reveal masses of biliary pigment. There is an increase in the excretion of urea and uric acid (Hofmeier).⁷ The stools are unaltered in character. The pulse, respiration and temperature of the

¹ Zeitschr. f. Kinderh., Orig., 1913, IX, 208.

² Zeitschr. f. Kinderh., Orig., 1913, IX, 198.

³ Amer. Jour. Dis. Child., 1912, III, 304.

⁴ Gerhard's Handb. d. Kinderkr., IV, 2, 691.

⁵ Klinik d. Leberkrank., 1858, I, 199.

⁶ Arch. f. exper. Path. u. Therap., 1885, XIX, 34.

⁷ Zeitsch. f. Geburtsh. u. Gyn., 1882, VIII, 287.

child are entirely unaffected, and the liver and spleen are not enlarged. Regarding the influence of the disease on the general health and nutrition there is some dispute. The majority of statistics certainly indicate that there occurs a greater loss of weight and a slower regain of it when icterus is present, but it is uncertain whether the slow gain is not rather the cause than the result of the icterus.

The duration is about 3 or 4 days in the milder cases. In the severer forms it may be 2 weeks or more before all traces of the icterus have disappeared, but in these long-continued cases it is always questionable whether the condition does not depend upon some other cause.

Diagnosis.—The distinction is to be made between icterus neonatorum and the various forms of symptomatic icterus. This is to be done in part by the presence of other symptoms in the latter condition. Then, too, in symptomatic icterus the urine is more liable to be visibly discolored by the bile-pigment. In cases of decided obstructive jaundice the stools show an absence of bile. A very slight degree of discoloration of the skin speaks rather for icterus neonatorum, while a late development of it indicates some other cause. Yet an early diagnosis is often impossible. This is especially true in slight cases of catarrhal icterus due to duodenal catarrh; and still more in the instances in which icterus neonatorum happens to be combined with other diseases of the new born; such, for instance, as sepsis. Congenital obliteration of the bile-ducts gives rise to an intense icterus developing very promptly after birth and persisting. Certain severe cases of congenital icterus will be discussed later. (See Digestive Diseases, p. 836.) Such infants are distinctly ill, and the icterus is accompanied by other signs, sometimes of a hemorrhagic nature.

Treatment.—Nothing is needed, and nothing can be done for the ordinary average case. Time will cure it. The ordinary care should be taken to keep the functions of the organism in good order.

CHAPTER VII

ASPHYXIA NEONATORUM

The term Asphyxia, or Suffocation, as applied to the new born indicates a condition in which, although the heart's action continues, the supply of oxygen to the blood ceases, and respiratory movements after birth are either absent or insufficient to overcome this lack.

Etiology.—The disease may either be (1) of *intra-uterine* or (2) of *extra-uterine* origin, the latter being much less frequent. The distinction depends on whether or not the circulation through the placenta has been interfered with.

(A) **INTRA-UTERINE ASPHYXIA.**—This develops before or during birth, and is due to some interruption of the normal interchange of gases in the placenta. Among maternal causes which interfere with the placental circulation may be mentioned excessive uterine contraction, unduly prolonged labor, uterine hemorrhage, and severe complicating illness or the death of the mother. On the side of the child are such factors as detachment of the placenta, compression of the brain interfering with the action of the heart, and compression of the umbilical cord. Efforts at intra-uterine respiration occur in most cases as a result of the undue stimulation of the respiratory centres; but whether this stimulation is the result of

the overloading of the blood with carbonic dioxide or of the deprivation of oxygen is not yet certainly determined. The consequent filling of the respiratory passages with fluid increases the danger to the infant after birth has occurred. In cases, however, where the asphyxia has developed slowly, the respiratory centres have sustained a paralyzing action, and efforts at breathing have not taken place.

It is evident that the likelihood of the occurrence of intra-uterine asphyxia must increase with the duration of labor, especially of its second stage. The statistics of Veit¹ show very strikingly that the mortality from asphyxia after a second stage of 4 hours or more was over 3 times as great as when it had lasted but 1 hour.

(B) **EXTRA-UTERINE ASPHYXIA** (*Atelectasis Pulmonum*).—The child is born without any evidence of asphyxia, but develops it soon after birth from some of the numerous causes which interfere with the gaseous interchange. Among these may be mentioned interference with the access of air, as by maternal discharges or unruptured membranes; malformation of the diaphragm; intra-uterine pneumonia or pleural effusion interfering later with the action of the lungs; malformation of the lungs; severe injuries to the brain at the moment of birth, which afterward affect the action of the respiratory centres; malformation of the heart, which renders the carrying of oxygenated blood impossible; etc. Premature birth is a very potent cause of extra-uterine asphyxia, the active factor being the general feebleness of the child, the weakness or imperfect development of the respiratory nerve-centres, or a similar condition of the muscles and bones of the chest-wall or of the lungs preventing satisfactory pulmonary or thoracic expansion.

Pathological Anatomy.—The lesions found are those characteristic of suffocation. The blood is fluid and of a dark color. The heart, especially the right chambers, is overfilled with blood. The blood-vessels in general are distended, and all the internal organs congested. The liver is of a dark bluish-red color. Numerous punctiform or larger hemorrhages may be found in nearly any of the organs of the body. Bloody serous fluid is present in the serous cavities. There is often the escape of a considerable amount of blood into the intestines.

If the child has made attempts at inspiration while still in the uterus, mucus, bloody amniotic fluid or meconium may be found in the larynx, trachea and larger bronchi; less often in the finer bronchi and the alveoli. The lungs are very dark red, heavy, much congested and of uniform atelectatic appearance when there has been no entrance of air. This intense congestion is evidence that intra-uterine efforts at respiration have probably taken place. If air has entered to some extent, either through the infant's own efforts or as a result of the employment of artificial respiration, small scattered areas of distended pulmonary tissue will be visible, especially in the upper lobes.

Symptoms. (A) **INTRA-UTERINE ASPHYXIA.**—In asphyxia of intra-uterine origin certain symptoms discoverable before birth make the diagnosis very probable. There occurs in the intervals between the labor pains a retardation of the fetal heart-sounds. Finally, if delivery does not occur, the sounds grow more irregular, rapid, and weak, and then cease altogether. A suspicious, although not entirely diagnostic, symptom in vertex presentations is the discharge from the maternal vagina of meconium which has just been passed by the infant, as a

¹ Monatssch. f. Geburtsk., 1855, VI, 112.

result of the increased intestinal peristalsis that asphyxia produces. The intra-uterine movements of the child may become more active. Exceptionally in breech presentations, convulsive movements of the body may be noticed. Sometimes, too, intra-uterine efforts at respiration may occasionally be detected by the finger inserted into the infant's mouth.

In the case of new-born healthy children respiration, followed by a vigorous cry, begins at once, or at the most after a very few seconds. In asphyxiated children, on the other hand, either no respiratory efforts at all are noticed after birth, or only imperfect and intermittent ones. The body is motionless and the child appears to be dead except for the continued action of the heart.

Two degrees of asphyxia of intra-uterine origin are observed: a milder form, *asphyxia livida* and a severer form *asphyxia pallida*. The symptoms, prognosis and treatment of the two are very different.

1. Asphyxia Livida.—In this milder form the skin is dark bluish-red in color, the heart's action is strong although decidedly slow, and the pulse in the umbilical cord is full and strong and the tension high. The conjunctivæ are injected, and the face turgid. Respiratory efforts are absent or occur only occasionally, and at first very superficially, and are attended by a contortion of the face. Coarse rales are audible in the lungs. Stimulation of the skin causes an energetic inspiratory effort, and the finger introduced into the mouth for the purpose of cleansing it produces attempts at vomiting or swallowing. A very characteristic symptom of *asphyxia livida* is that the muscle-tonus is preserved; *i.e.* although the child is motionless, yet it is not absolutely flaccid when it is lifted.

2. Asphyxia Pallida.—In this variety the skin is pale and corpse-like, and the heart's action very weak and usually rapid. The vessels in the cord appear to be empty, and the pulse there is entirely absent or extremely feeble. Generally there are no true respiratory efforts, and any occasional attempts at inspiration which may occur seem to depend entirely on the action of the diaphragm, the thorax moving not at all, and no grimace of the face attending them. No rales can be heard; this showing that the efforts at inspiration have been absent or entirely futile. Stimulation of the skin is without result, and the finger in the pharynx produces no reflex movements of its muscles or of the palate. The great characteristic of this grade of asphyxia is complete loss of muscle-tonus. The child is absolutely limp, the jaws fall, the head drops completely in some direction if the child is lifted, and the anus is open.

(B) EXTRA-UTERINE ASPHYXIA.—In asphyxia of extra-uterine origin the symptoms vary somewhat, depending upon the cause. The skin is usually dark reddish-blue. The heart's action is strong, and the vessels of the cord are filled with blood. The pulse, however, is not at first slow, and although it later becomes so, it finally commonly grows rapid again. This distinguishes the condition from intra-uterine asphyxia. Respiration is wanting or occasional only, and is not attended by rales. This lack of rales shows the absence of fluid from the respiratory tract, an evidence that the condition is extra-uterine in nature. In asphyxia in premature infants respiration may continue irregular and very superficial for days, and is almost entirely diaphragmatic. The children lie for the most part with eyes closed, motionless and somnolent, and make no sound or only occasionally utter a feeble cry. The face is dark red and somewhat swollen; edema of the extremities and scrotum develops; the temperature is subnormal; there is loss of weight. In marantic infants

who have passed the age generally allotted to the new born, as well as later in badly rachitic subjects, asphyxia is of not infrequent occurrence. It is the result of atelectasis, and its symptoms are those of the extra-uterine form developing earlier in life.

Course and Prognosis.—Unless treated there is probably little tendency for cases of asphyxia to recover. *Asphyxia livida*, when not excessive, will nearly always yield quickly to appropriate treatment. In the course of a few seconds or possibly minutes respirations become more frequent and effective, and are finally succeeded by loud crying. Many children, however, pass from the milder form into the severer degree. The result in *asphyxia pallida* is always more doubtful. The duration is variable. Some children die almost immediately, while others may continue for hours without any apparent change; or, after improvement begins, may relapse if treatment is not persisted with. If recovery ensues intermittent and occasional respirations begin and are perhaps followed by a slight cry, and finally satisfactory breathing is established. The longer the duration of the second stage of labor, the worse is the prognosis. The prognosis is graver, too, in proportion to the weakness of the pulse or of the heart-sounds. The presence of any complication, such as intracranial hemorrhage or acute fatty degeneration, likewise makes the prognosis unfavorable. Yet even in the severer grades of intra-uterine asphyxia the majority of infants will recover. Even those who have seemed hopeless, and who for hours have appeared to be dead may still survive. Asphyxia of extra-uterine origin generally runs a rapid course if dependent upon some malformation or some disease of the respiratory apparatus. When the result of premature birth, it may continue, as stated, for days.

Regarding the after-effects of the disease upon a child who has survived, it has been maintained that long-continued severe asphyxia is liable to produce idiocy or paralytic conditions. Although it seems more probable that such disorders are the result of some injury to the brain at birth, of which the respiratory condition was but a symptom, yet it is possible that the congestion accompanying the asphyxia may have produced the breaking of blood-vessels within the cranium, and thus have become the first cause of later nervous or mental disorders. It may happen, too, that aspiration-pneumonia may develop in an infant after its recovery from asphyxia.

Diagnosis.—The milder grade of asphyxia may be confounded with *compression of the brain* by hemorrhage. Meningeal hemorrhage may indeed produce symptoms resembling those of asphyxia very closely, and as the latter condition may be one of the symptoms of the former, the object of diagnosis is to determine whether or not asphyxia is actually present, and, if so, whether it depends on an intracranial compression. Compression produces slowing of the pulse and irregularity of respiration. If a child exhibits at birth the symptoms of the milder degree of asphyxia and, instead of responding quickly to treatment, does so not at all, or but temporarily or imperfectly, while the slowness of the pulse persists, the existence of a complicating meningeal hemorrhage is very probable. This is true also if the child is born without evidence of asphyxia, but soon passes into this condition, with slow pulse, slow irregular respiration, coma, and bulging fontanelle. In other cases there is marked asphyxia from the beginning and the diagnosis must for a time remain in doubt. The presence of paralysis renders the diagnosis of hemorrhage positive. The history of the case aids in the recognition of compression; as, for

instance, when the labor has been unduly prolonged, the pelvis narrow, or forceps applied.

Intense anemia resulting from severe hemorrhage may simulate asphyxia pallida closely. The presence of rales, if a respiration occurs, is an indication that intra-uterine efforts at breathing have taken place and that asphyxia exists. The history is of the greatest diagnostic value, for only the tearing of the umbilical cord during birth can produce a hemorrhage which is sufficient to account for so great a degree of anemia.

Asphyxia may be complicated by, or be the only symptom of, *acute fatty degeneration of the new born*. In such cases the issue is fatal, and the microscopic examination makes the diagnosis clear.

Treatment. (A) ASPHYXIA OF INTRA-UTERINE ORIGIN.—The condition should be prevented as far as may be by employing such measures as will obviate unduly prolonged delivery and in every other respect maintain the labor in as normal a condition as possible. Evidences of asphyxia developing during labor must be carefully watched for. The subject is treated of in works upon obstetrics.

Whatever the degree of asphyxia present after the birth of the infant the first indication is to free the respiratory passages as far as possible. A finger should be introduced into the mouth and pharynx in order to remove any mucus or fluid present. The child may also be suspended with head downward for a moment in order to favor discharge of fluid, and to produce a congestion and stimulation of the respiratory centres, and the body be slapped smartly with the open hand. In the case of **asphyxia livida**, if well marked, the cord may be severed at once, and $\frac{1}{2}$ ounce (15) or less of blood allowed to escape. The child may then be slapped with a cold, wet towel or plunged into cold water in order to stimulate respiration by cutaneous irritation. It must then immediately be put into a hot bath. This procedure, involving the alteration of hot and cold water, may be quickly repeated as often as necessary. Efforts at respiration are generally produced by the cold water. These are attended by fresh accumulations of mucus in the throat necessitating repeated cleansing of this in the manner mentioned. Sometimes a single application of cold will suffice. In other cases we must continue perhaps a half hour before all evidences of abnormal color have disappeared, and the infant is breathing well and crying lustily.

In **asphyxia pallida**, or in those cases of asphyxia livida in which the treatment described does not quickly produce some decided efforts at respiration, valuable time is lost by employing it. In the latter form of the disease the child has passed the stage where cutaneous stimulation can produce any effect whatever, and in the former the permitting of a flow of blood from the cord can do only harm. The cord should be ligated and cut promptly, and artificial respiration commenced at once. Whatever means are employed an artificial *expiration* should be sought first, in order to remove aspirated fluid from the respiratory tract. If inspiration occurs first the fluid is drawn still further into the lungs. The method of Schultze is that most in vogue.¹ The physician grasps the child with both hands in such a way that his thumbs rest loosely on the front of the child's thorax, his first fingers pass from behind into the axilla, and the remaining fingers are spread over the back of the child. The infant's head rests against his forearms and hands. Standing with knees somewhat apart, and having the child hanging feet downward and face forward, he now swings the body forward and upward, exercising no com-

¹ Der Scheintod d. Neugeborenen, 1871.

pression with the fingers, until his arms are somewhat above the horizontal position. The swing now ceasing, momentum carries the lower part of the child slowly forward, throwing the legs up and over toward the physician, while the head hangs down. The trunk becomes in this way strongly flexed, and the abdomen and chest consequently compressed, the weight of the child resting against the physician's thumbs. This pressure causes an enforced expiration, and the aspirated fluid may be driven from the mouth and nose. After a pause of a few seconds the child is again swung downward into the original position. All pressure being thus removed from the chest, an enforced inspiration follows. In a few seconds more the movements are repeated, and so on for 6 or 8 times in the course of a minute or less, after which the child is placed in a tub of warm water for a few minutes. If no natural respiratory movements have commenced, the whole process is repeated (Fig. 47).

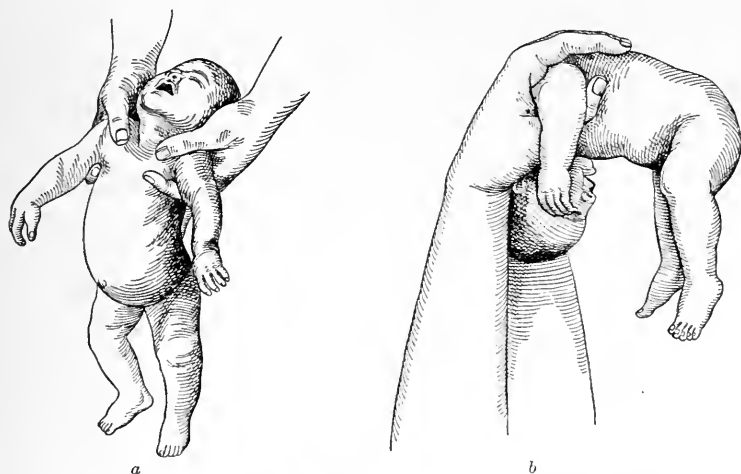


FIG. 47.—SCHULTZE'S METHOD OF ARTIFICIAL RESPIRATION.
(a) Inspiration; (b) expiration. (*B. C. Hirst, Obstetrics, 6th Ed. 958.*)

It is important that the inversion of the child in the production of expiration should occur slowly, in order that the flexion of the spine may take place in the lumbar region and not above this. The first part of the swing upward is quickly made, the latter part more slowly. The neck must always be kept from flexing in order to permit free entrance of air. Any compression by the hands must be carefully avoided at every stage. The movements, though performed thoroughly, must always be done gently and skilfully. The method requires some practice to be carried out properly. If the swinging movements have succeeded in starting respiration and increasing the power of the heart's action, the case has now become one of the milder degree of asphyxia, and cutaneous stimulation may be tried. If it has not succeeded artificial respiration must be used again and again. It should be persisted with as long as the heart continues to beat, even for hours if necessary.

It is, in fact, very important not to abandon too soon this or any other method of resuscitation which is employed. A few efforts at respiration by the infant, or a single cry, do not constitute recovery, as relapse is very liable to occur. Easy respiration must be fully established, the

normal color must return, the extremities move, and the child cry well and open its eyes before the physician may feel content. Very careful supervision is necessary after respiration seems established. Small doses of alcoholic and other cardiac stimulants are useful, as well as careful protection against chilling by the use of cotton wrappings and hot-water bags.

Weakly infants with great debility of the heart's action may not well tolerate the Schultze's treatment, or the chest walls may be so yielding that inspiration does not occur. In other instances the presence of such injuries as fractures, especially of the arms or clavicles, interferes with the use of the method, and always there is the difficulty in avoiding chilling. In place of it the direct inflation of the lungs may be tried. Mouth-to-mouth inflation is an old method, which may be employed in emergency, a clean towel being placed between the mouth of the child and that of the physician and only the first part of the expired air from the physician blown into the child. The nostrils should not be compressed. If this does not succeed a soft rubber catheter may be passed into the larynx, and, after fluid aspirated by the child has been withdrawn by suction by the mouth or by a rubber bulb attached to the tube, air may be forced into the lungs and again drawn out, aided by pressure of the hand upon the thorax. This should be continued, using fresh air at each inflation until respiration is established. The method is not without objections, for not only is it difficult to pass the tube into the larynx instead of the esophagus, but too great pressure may easily over-distend the lungs and produce emphysema or even rupture of the vesicles. A more scientific modification of this is the furnishing of a continuous supply of air, as used in animal experimentation, and the employing of a manometer to guard against too high a pressure.

Numerous modifications of or substitutions for Schultze's method have been proposed, some of them antedating it. Among these may be mentioned those of Marshall Hall¹ and of Sylvester² as used for the recovery of persons from drowning, and the method of resuscitation recommended by Laborde.³ This latter consists in grasping the tip of the tongue, wrapped about with gauze, and making rhythmic traction on it 8 to 12 times a minute. It is a powerful awakening of respiratory efforts if there is any reflex excitability remaining in the nervous supply of the tongue. Its advantage over Schultze's method is that it can be used while the child is kept in the warm bath. According to Dew's method⁴ the left hand of the physician grasps the infant by the back of the neck and the right hand the knees, the body and thighs resting upon the palms. The lower extremities and abdomen are now flexed upon the thorax and head producing expiration, and the body and head then extended into a backward curve to bring about inspiration. This seems to be practically identical with the method of Byrd,⁵ which consisted in letting the child lie with its back upon the palmar surface of the physician's two hands. The body is then flexed, bringing the head and feet close together and forcing the air from the chest. A movement of over-extension in the opposite direction tends to produce inspiration. A similar procedure is recommended

¹ *Lancet*, 1856, II, 124.

² *Brit. Med. Journ.*, 1858, 576.

³ *Les tractions rythmiques de la langue*, 1897.

⁴ *Koplik, Dis. of Inf. and Childh.*, 1910, 196.

⁵ *Baltimore Med. Journ.*, 1870, I, 646.

by Ssokolow¹ (Fig. 48), the head being allowed to flex toward the spine during the extension of the body. These methods have the advantage that the infant can be kept in the warm bath much of the time. Minkévitch² places the infant on its back, with the hand of the physician in each axilla, and alternately bends the trunk forward between the separated legs, and extends it again into the horizontal position, while Rosenthal³ flexes the knees upon the breast with the infant similarly placed. Prochownik⁴ advises that the child be held inverted while an assistant



a



b

FIG. 48.—METHOD FOR INDUCING ARTIFICIAL RESPIRATION IN THE NEWBORN.
(a) Inspiration; (b) expiration. (After Ssokolow, *Monatsschr. f. Kinderh., Orig.*, 1911, X, 459.)

alternately compresses and releases the thorax; Zangenmeister⁵ recommends forced inhalation of oxygen through a tracheal catheter fitted with a bulb; and La Rue⁶ claimed good results in re-establishing cardiac action by massage of the heart by the thumbs over the precordial area.

¹ *Monatsschr. f. Kinderh., Orig.*, 1911, X, 457.

² *Semaine méd.*, 1902, XXII, 372.

³ *Therap. Monatsh.*, 1893, VII, 55.

⁴ *Centralbl. f. Gynäk.*, 1894, XVIII, 225.

⁵ *Centralbl. f. Gynäk.*, 1903, XXVII, 1162.

⁶ *Pediatrics*, 1914, XXVI, 126.

(B) ASPHYXIA OF EXTRA-UTERINE ORIGIN.—Treatment is of little avail where the asphyxia results from anomalies of respiration or of circulation, or from intra-uterine diseases. When it depends upon the existence of premature birth there become necessary certain modifications of the methods already detailed. Not only is the relief of the asphyxia required, but that treatment also which has been described as appropriate for premature birth (p. 255). The removal of aspirated fluid from the mouth is not usually needed, since intra-uterine efforts at respiration have not taken place. When the case is one unsuited for, or not benefited by, cutaneous stimulation, artificial respiration must be attempted. However, the weakness of the child and the lack of elasticity of the chest-wall, together with the great need for maintaining bodily heat, render Schultze's method in many cases inapplicable or unavailing, and some of the procedures to be preferred which permit of the constant maintenance of bodily temperature in a warm bath. The very great liability of these cases to relapse is not to be forgotten. Close watching is required, and if the tendency appears, warm baths and cutaneous friction should be employed several times daily in order to interrupt the somnolence and produce vigorous crying. Days may pass before either the safety of the child is assured or death takes place.

CHAPTER VIII

PULMONARY ATELECTASIS IN THE NEW BORN

Etiology.—The name denotes a persistence of, or a return to, the unexpanded fetal condition of the lung. It is peculiarly a disease of the new born, although under certain circumstances it may develop in older subjects. This acquired atelectasis will be considered later (Vol. II, p. 94). In some cases the infants are born asphyxiated, respiration is absent or incomplete, and the lung never expands properly even under the influence of artificial respiration. The asphyxia and the atelectasis may then be regarded as due to the same cause. (See Asphyxia, p. 276.) In other instances, especially in premature children, or those who are weakly from other reasons, respiration is established for a time and there is at first no evidence of asphyxia. Soon, however, the poorly distended lungs become gradually more and more collapsed, owing to the weakness of the child and the yielding character of the thorax, which make satisfactory respiration difficult or impossible. Asphyxia then develops as the result of the atelectasis.

Pathological Anatomy.—In complete atelectasis, where the infant has never breathed, the lungs are dark-red or blue-red, small, very vascular, entirely collapsed, firm to the touch, do not crepitate, and sink in water. They are capable, however, of being forcibly inflated without difficulty, thus distinguishing the condition from pneumonic consolidation. If the lungs have been partially inflated during life, either through respiratory efforts of the infants or through attempts at artificial respiration, post-mortem examination shows small scattered areas of normally distended or emphysematous pulmonary tissue, characterized by the lighter color and the evident presence of air. These are oftenest seen in the upper portion of the lung or at the anterior edges. Where life has continued some weeks the anterior parts of the lungs may

be found entirely distended or even emphysematous, while the posterior portions, especially the lower lobes, are completely atelectatic, or perhaps the surface seems normal but the portions beneath it are collapsed and the lung is hard. There is a striking contrast between the areas of healthy or distended lung and the depressed, dark, atelectatic portions. Both lungs are generally involved, although not necessarily equally so. Quite frequently more or less hypostatic pneumonia is combined with the atelectasis. In some cases one area may be pneumonic and another atelectatic. Other lesions are those described under Asphyxia.

Symptoms.—In the cases in which the children are asphyxiated at birth, and either show little or no tendency to recovery or relapse promptly, the symptoms of atelectasis are those already described under Asphyxia. In those in which the atelectasis develops later, being dependent upon the excessive weakness of the infant, or in which asphyxia, although present at birth, has apparently been recovered from, the symptoms of atelectasis are characteristic. The pulse is weak and slow; the respiration is rapid, irregular and shallow, and attended by evident retraction of the intercostal spaces and the lower portion of the thorax at the insertion of the diaphragm. The infant is somnolent and lies generally with eyes closed, crying but little and never loud, and making feeble, if any, efforts to suck. The temperature of the body is persistently or only at times subnormal. The physical examination of the lungs generally gives a somewhat impaired percussion noted over the lower posterior portion of the chest. Sometimes, however, no distinct alteration of note can be discovered if numerous inflated areas of pulmonary tissues are present. The respiratory murmur is feeble, with fine râles at times, but as a rule no bronchial breathing is discovered.

This condition may continue for weeks. Recovery may take place after several relapses, or death may occur suddenly, often with convulsions and without there having been any very positive sign which indicated the actual state of the lungs.

Prognosis.—When the disease is present at birth and is accompanied by asphyxia livida, and where no congenital malformations or other pathological conditions are present the prognosis is generally favorable. When atelectasis occurs in premature infants the result is more uncertain on account of the constant danger of relapse. Yet very great care and watchfulness will often succeed in saving life.

Diagnosis.—The disease is to be distinguished principally from the hypostatic pneumonia which is liable to develop in weakly infants. The absence of fever and of bronchial respiration constitute the chief diagnostic evidences against pneumonia.

Treatment.—Although varying with the nature of the causes and symptoms, treatment is practically identical with that recommended for asphyxia. Cutaneous stimulation, artificial respiration, the maintenance of the bodily temperature, and the frequent administration of nourishment are indicated. (See *Premature Birth and Asphyxia*, pp. 252 and 276.) It must never be forgotten that atelectatic infants must have an abundance of fresh warmed air and must not be allowed to lie too long in one position. They should be roused from their somnolent condition at frequent intervals and carried about cautiously; and be given massage, warm baths and similar measures to stimulate both circulation and respiration.

CHAPTER IX

CONGENITAL ASTHENIA

In this category belong the infants born with a power of resistance to deleterious influences and a capacity of thriving under ordinary circumstances much below normal. The condition is different from that of premature birth, although often associated with it. Premature birth is doubtless the most frequent cause, but not every case of prematurity exhibits debility, while, on the other hand, infants born at full term may be suffering from asthenia. Such cases of congenital asthenia may be the result of prenatal influences such as prolonged illness of the mother, and particularly such conditions as syphilis, tuberculosis, alcoholism, and the like. In some of these the baby, although born at term, is physically premature, the maternal condition having interfered with intra-uterine development. In other cases the fact that the infant is one of multiple births accounts for the asthenia. The condition is much the same whatever the cause, and the symptoms and treatment are fully considered in the chapter on Premature Birth. There is often a difference, however, in the prognosis. If a prematurely born infant with asthenia dependent upon no maternal disease can be maintained alive until its organs reach the power of functioning properly, the prognosis is good; whereas the infant with constitutional debility, perhaps depending upon prenatal influences, may show little increase of energy as time passes.

CHAPTER X

DISEASES OF THE UMBILICUS

Affections of the navel are among the common pathological conditions of the new born. In 2603 births Porak and Durante¹ found some abnormality in 832 or 32.6 per cent. although this was of an important nature in but 333; viz., 12.8 per cent. All the severer forms have become much less frequent since antisepsis has been practised more perfectly.

The various umbilical disorders, with the exception of hernia which will be discussed later (p. 790) may be subdivided as follows:

DELAYED HEALING OF THE UMBILICUS

(Excoriation; Blennorrhœa; Umbilical Ulceration, etc.)

Under this title Runge² properly groups several minor affections of the umbilicus which, although often described as distinct conditions, are yet clearly allied.

In place of skinning over with epithelium a few days after the fall of the cord, the umbilical wound sometimes projects slightly and becomes irritated by the dressing applied, constituting the so-called *excoriatio umbilici*. If a flat, red surface is visible, resembling mucous membrane, and secreting pus more or less abundantly, *blennorrhœa of the navel* is spoken

¹ Arch. de méd. des enf., 1905, VIII, 465.

² Krankh. d. ersten Lebenstagen, 1893, 71.

of. Should the process extend in area or in depth a condition of genuine ulceration is produced with a granulating surface, sometimes covered with necrotic tissue and constituting an *ulcus umbilici*. This ulcer may develop a false membrane, and is then entitled *croup* or *diphtheria of the navel*, without the process necessarily being of a truly diphtheritic nature.

Etiology.—The causes of this delayed healing are various. There is a certain normal variation in the falling of the cord and the healing of umbilical wounds. An average time for the separation of the cord is the 5th day, and for the healing of the wound the 12th to the 15th day. In strong children the degree of inflammation is liable to be more intense than in weakly ones, the cord falls sooner, and the wound heals more rapidly. It is only when the inflammation increases after the cord has separated that the process can be called abnormal. In some cases local irritation by the dressing is the cause of the inflammation. Lack of cleanliness is another factor. In many instances a local infection is without doubt present, and this is probably nearly always the case when ulceration develops. It is also likely that infection has taken place if decided increase of redness and secretion is combined with a much delayed separation of the cord.

Symptoms.—No general symptoms attend these different forms of delayed healing of the umbilical wound, and no danger to life exists provided they remain purely local. Even ulceration of the umbilicus is without involvement of the general health unless it has lasted a considerable time. Where there are decided constitutional symptoms accompanying the umbilical lesions described infection has certainly extended beyond the umbilicus.

Treatment.—Inasmuch as the persistence of the umbilical wound continues the liability to septic infection, treatment is necessary. As a prophylactic measure the stump of the cord must be kept as dry as possible in order to hasten mummification and separation. Should there be delay in healing after the cord has separated, dressings with some antiseptic powder are to be preferred. After a thorough cleansing with water followed by careful drying, powdered boric acid may be applied in a thick layer, or a mixture of salicylic acid and starch or talc in the proportion of 1:5, and this covered with a wad of absorbent cotton and a bandage. Washing with hydrogen dioxide, and painting with a 1 per cent. solution of nitrate of silver are often also of service. The dry dressing should be renewed once or twice daily.

FUNGUS OF THE UMBILICUS

(Granuloma; Sarcomphalos; Umbilical Polypus)

After the separation of the cord an abnormal growth sometimes develops at the navel. This may be hidden by the overlying skin, or may project as a round, red mass the size of a pea or larger, exhibiting a granular appearance on close inspection. The surface of the mass is moist, discharges sero-purulent fluid, and bleeds slightly when irritated. Unless treated the growth may persist for months. The general health of the infant is unaffected.

Anatomically it is composed of granulation tissue which has developed upon the point of attachment of the cord. The condition can always be recognized if the folds of the skin about the navel are pulled apart in a way to render the interior visible. It is to be distinguished only from a persistent Meckel's diverticulum (p. 810). The tendency to recovery

unaided is slight, and the umbilical wound cannot heal while the fungus is present. Treatment consists in the application of astringents, preferably nitrate of silver, followed by a dressing of powdered boric acid. This may be repeated every few days if necessary. If the fungus is of considerable size it may be ligated. When exuberant granulations have disappeared an antiseptic dressing may be applied until the wound becomes covered with epithelium.

OMPHALITIS

Periomyelitis

Etiology.—The term is used to designate a phlegmonous inflammation of the navel and surrounding tissues. It is not of frequent occurrence, Hennig¹ finding but 12 cases among 7000 sick infants. The cause is a pyogenic infection, to which uncleanness and lack of proper care of the navel have predisposed.

Symptoms.—The disease begins usually in the 2d or 3d week of life, after separation of the cord and as a sequel to a delayed healing of the umbilical wound, which may exhibit ulceration with secretion of pus. The skin around the navel is swollen, red, hot, shining and projecting, with a disappearance of the normal folds. The subcutaneous tissue is infiltrated and hard, and the slightest pressure causes intense pain. The inflammation may remain comparatively superficial, or may extend in depth, even involving the peritoneum. It may be confined to the neighborhood of the navel, or may attack the greater part of the abdominal wall. In some such cases it is very probable that an erysipelatos infection has been added to the omphalitis, or has been present from the beginning. The general condition of the infant is always affected. There is restlessness, loss of appetite, and fever. Pain is a prominent feature, and on this account there is little movement of the abdomen or lower limbs, the thighs are generally held rigidly flexed on the abdomen, respiration is superficial, and loud crying is avoided. The emptying of the bladder and of the bowels is also painful.

Course and Prognosis.—The duration and course of the disease is variable. In the mild cases the exudate is rapidly absorbed. A few pustules or quite small abscesses may form in the vicinity and recovery is complete in a few days. The severe cases may last for weeks, the infiltration being slowly absorbed, or an abscess developing and discharging. The prognosis is always dubious. Those cases generally recover in which the disease spreads but little. Where the process is intense the prognosis is grave. The more quickly the severer cases advance to the formation of abscess the better for the child, since the duration of the inflammation and the consequent exhaustion are thus curtailed. The development of a complicating gangrene or the extension of the lesion to the peritoneum makes the prognosis very unfavorable. If the inflammation involves the umbilical vessels general septic infection will probably follow.

Diagnosis.—This is as a rule rendered evident by the pain and the infiltrated, hard, prominent umbilical region. The formation of an abscess is shown by the presence of fluctuation. Erysipelas is generally distinguished by the rapid spreading of the characteristic color. Yet when erysipelas exists as a complication, the diagnosis of the existence of a primary omphalitis becomes most difficult.

¹ Gerhardt's Handb. d. Kinderkr., II, 131.

Treatment.—This is first of all prophylactic. The navel must be kept aseptic and free from irritation. If omphalitis has already commenced, the umbilical wound should be cleansed carefully and then powdered thickly with boric acid, iodoform, salicylic acid and starch (1:5), or other antiseptic powder. If there is much infiltration of the tissues and abdominal pain, the inflamed area may be covered with a warm, wet antiseptic dressing. A diluted ichthylol ointment, 5 to 10 per cent. is sometimes useful. Any abscesses which form should be incised early. Attention must be given to the nourishment of the infant, forced feeding being used if necessary, and stimulants being required in many cases. The bowels should be emptied by injections, as the child cannot make any effort at pressing.

GANGRENE OF THE NAVEL

Etiology.—Gangrene may be a sequel to severe cases of omphalitis, ulcer, or inflammation of the umbilical vessels. It is now very rare as a purely local affection, since better antiseptic precautions are observed; but is still seen, though infrequently, as a secondary manifestation of a general septic infection. It may exceptionally follow severe general diseases, especially diarrhea of a choleraic nature, even in children over a month old and previously healthy. The existence of great debility, as in cases of premature birth, favors its development. An especially potent factor is lack of cleanliness about the umbilical wound.

Pathological Anatomy and Symptoms.—The inflammation already present develops into a greenish or black offensive mass surrounded by a red areola, and the edges of the wound become discolored and break down, causing a more or less rapid loss of substance, either in area or in depth. In the latter case the process may extend to the peritoneum, and even into the intestine, producing perforation and fecal fistula. If the spreading is toward the periphery the greater part of the superficial abdominal wall may be destroyed, involving more or less the muscular layer and even extending to the bladder. Severe hemorrhage may occur if the umbilical vessels are involved. General sepsis may be produced by way of the vessels or of the peritoneum. The constitutional symptoms attending gangrene are always severe. There are great prostration with coldness of the extremities, quick and weak pulse, and little or no fever. Rapid collapse is frequent. Occasionally the process is not so serious, the gangrene may not extend far, and, the reactive inflammation producing pus the dead tissue is thrown off and the cavity fills with granulations. The diagnosis offers no difficulty.

Course and Prognosis.—The duration varies, depending upon the resisting power of the infant and the swiftness of the spread of the disease. The average duration of fatal cases, according to Fürth¹ is about 5½ days, and of those which recover about 22 days. The prognosis is exceedingly bad. If the diseased area is small, the infant's strength good, and reactive inflammation soon sets in, recovery may follow, but the mortality reaches over 85 per cent. (88.48 per cent., Fürth). Absence of the surrounding areola, involvement of the peritoneum, severe hemorrhage from the umbilical vessels, and development of general sepsis render death almost inevitable. Gangrene arising as an affection secondary to constitutional conditions and other diseases is uniformly fatal.

¹ Wiener Klinik, 1884, X, 331.

Treatment.—This consists in supporting the strength by means of abundant nourishment and powerful stimulation, the maintaining of the temperature, the limiting the spread of the disease, and the favoring of the casting off of the dead tissue and of the development of a reactive inflammation. Warm wet antiseptic dressings may be applied. The early employment of the thermo-cautery may be efficacious. After the gangrenous tissue has separated iodoform or ichthyol may be used.

UMBILICAL ARTERITIS AND PHLEBITIS

Etiology.—Both of these conditions are of comparatively unusual occurrence. The investigations of Runge¹ show that inflammation of the arteries is far more common than that of the vein. In 55 cases of disease of the vessels the vein was found affected but once while in all instances the arteries were involved. Of all affections of the navel arteritis appears to be much the most frequent cause of death. Phlebitis, too, usually leads to general fatal septic inflammation. The cause of disease of the umbilical vessels is always an infection of the umbilical wound. It is most likely to occur before the stump of the cord has completely separated, since the granulations which develop later form to some extent a protection against infection. It may appear as an epidemic in lying-in institutions, although this has become very much less frequent since better methods have prevailed. The infection may be acquired from the lochial discharges of the mother, or be transmitted by the hands of the physician or nurse, infected umbilical dressings, or even apparently by the air of an infected room or the water used for bathing. Any irritation of the umbilical wound predisposes to it, as does omphalitis or gangrene of the navel. On the other hand the vascular infection may occur without the umbilical wound showing anything abnormal.

Premature infants seem especially predisposed to the disease. The presence of moisture and decomposition in the stump of the umbilical cord is more favorable to the development of organisms than the normal state of mummification (Cholmorogoff).² Various organisms have been found in the stump of the cord and in the umbilical wound in cases of infection of the vessels, among these being varieties of staphylococci, the streptococcus pyogenes, the bacillus coli, the pneumococcus, and the bacillus pyocyaneus.

Pathological Anatomy.—The process always begins as an infection and inflammation of the perivascular tissue, according to the statements of Runge,³ which becomes infiltrated and swollen. It next extends to the vessel-walls and a septic thrombus forms in the vessel. The disease usually remains local, resulting in suppuration in or about the affected portion of the vessel or between the abdominal wall and the peritoneum, and the final discharge of pus. In a smaller number of cases the process spreads by continuity along the perivascular tissues, the sepsis reaching the general system through the perivascular lymph channels.

On inspection at autopsy the diseased arteries are visible as thickened, stiff and discolored cords, surrounded by edematous infiltrated connective tissue. The vessels of the adventitia are abundant. Nearly always both arteries are involved either for a short distance only or

¹ Krankh. d. ersten Lebenstagen, 1893, 88.

² Zeitsch. f. Geburtsh. u. Gynäk., 1889, XVI, 16.

³ Loc. cit.

throughout their extent. They contain broken-down thrombi, partially greenish in color from the purulent changes, partially still bloody. The intima of the vessel is without the natural shining character and exhibits loss of endothelium. The lesions of the umbilical vein are identical with those of the arteries, and the process usually extends throughout its entire length up to the liver and even into it. The umbilical wound may appear perfectly normal or may exhibit the lesions of ulcer or of omphalitis. Even if the stump of the cord is mummified and still persists, foci of suppuration may be discovered about the periphery of the region of beginning detachment. Some of the lesions of a general septic infection may be present, such as have been described in considering Sepsis in the New Born. (See p. 258.) In the case of phlebitis the liver may be involved directly by continuity from the umbilical vein.

Symptoms and Diagnosis.—Characteristic symptoms of the disease, apart from the pathological lesions, are practically absent. When the process remains a local one, as is true of the majority of cases, the symptoms are chiefly local. If the portion of the vessel immediately beneath the navel is involved, crusts may appear upon the umbilical wound, or there may be discharged from it on pressure pus which clearly comes from beneath the surface. In the cases where a portion of the vessel more deeply situated is the seat of the lesion, the navel is of normal appearance. In some such instances the hard and thick arteries can be felt through the abdominal wall. Sometimes abscess forms above the peritoneum and may make its way in different directions. Moderate fever and other constitutional disturbances may be present, but frequently no general symptoms whatever are discovered; and it may well be that death occurs unexpectedly, and only at autopsy is an umbilical arteritis found. In the more severe cases, in many of which the vessels are involved throughout much of their extent and general sepsis follows, there is often no disorder of the navel visible, the symptoms are very uncharacteristic, and there are no positive means of recognizing the disease during life. Without any apparent reason, or with no previous evidence of illness, the infant becomes restless, loses appetite, develops irregular and often high fever, and passes rapidly into collapse. Less frequently the duration is more prolonged, and symptoms of grave constitutional disturbance are present. High fever and collapse alternate, and wasting becomes great. There is diarrhea and distended and tender abdomen. Intense icterus is constantly present in umbilical phlebitis if the condition is at all prolonged (Wiederhofer),¹ and is frequent in arteritis also. In neither case is there any reason to justify the diagnosis of extensive inflammation of the umbilical vessels unless the umbilical wound is distinctly diseased, in which event the suspicion is warrantable, although proof is absent. When evidences of diffuse general pyemia develop, the diagnosis of an umbilical arteritis or phlebitis is rendered still more probable, although not even then certain.

Complications.—Pneumonia is that oftenest seen, and may be the only one. It occurred in 22 of Runge's 55 cases. All the widespread local pyemic lesions of septic infection may occur as complications, among these empyema, peritonitis, subcutaneous abscess, nephritis, and the like. (See Septic Infection of the Newborn, p. 258.) Erysipelas may sometimes develop as a complication.

Course and Prognosis.—As we cannot discover the time when the disease begins the exact duration is uncertain. In nearly all instances

¹ Jahrb. f. Kinderheilk., 1862, V, 195.

it is short, ranging from a few days to several weeks. The greater number of deaths in Runge's cases occurred on the 8th day of life. The prognosis is always grave. Since the diagnosis can only be made with certainty at the autopsy, it is impossible to determine its fatality, but it is probable that recovery does occur in many instances if the process does not spread beyond the vessels. Should the sepsis become general death is almost inevitable.

Treatment.—Prophylaxis is the most important. Careful antiseptic treatment of the umbilicus is necessary from the moment of birth. The hands of the physician and nurse must be aseptic, the scissors, ligature and dressings likewise so, and precautions taken at all times against the access of germs. The child should be at once removed from the vicinity of the mother if she has any evidence of sepsis. The stump of the cord must be kept as dry as possible in order to avoid a moist decomposition, and the frequent renewal of antiseptic dressings may be necessary to accomplish this. The use of salicylated cotton with a powder of salicylic acid and talcum (1 : 5) is useful for this purpose. The employment of powdered gypsum on cotton has been recommended by Sutugin,¹ Cholmogoroff² and others as the best means of accomplishing this. Such a dressing should be used as will not shut out air too greatly, since rapid drying is hindered in this way. When the cord has separated the wound should be dressed with the powder of salicylic acid and talcum, or one of salicylic acid and starch, (1 : 5) or with boric acid or iodoform. If the navel is already diseased in any way the treatment indicated for the condition present must be employed in order to prevent the spread of infection to the vessels.

Treatment of the disease itself is rarely possible even could the diagnosis be made with certainty. The preservation of the strength by abundant nourishment and by the free use of stimulants is indicated. Complications are to be treated as they arise.

OMPHALORRHAGIA

(Umbilical Hemorrhage)

(See also Hemorrhage in the New Born, p. 263)

This, like melena, is not a distinct disease, but a symptom of different conditions, itself of enough importance to warrant separate consideration. It is customary to recognize 2 forms (*A*) *accidental*, proceeding from the umbilical vessels, and (*B*) *idiopathic* or *spontaneous* coming from the umbilical tissues.

Etiology.—(*A*) *Accidental Hemorrhage.*—Severe hemorrhage from the umbilical vessels may occur before the fall of the cord as a result of imperfect ligation of it. The ligature may have been too loose, or may have slipped, or have cut into the vessels. Under otherwise normal conditions hemorrhage from such causes will not take place except in the first 10 or 15 minutes of life. When, however, under the influence of well-developed asphyxia in otherwise healthy infants, or especially in the slow, unsatisfactory expansion of the lungs in premature infants, the blood is not drawn into the expanding lungs in a normal manner, there results a maintenance of the blood-pressure in the umbilical arteries, the muscle of the arterial walls fails to contract and close the lumen properly, and

¹ Wratsch, 1883, No. 44, Ref.

² *Loc. cit.*, 28.

the occurrence of hemorrhage is possible. In like manner gangrene of the umbilicus or imperfect mummification allows the vessels to open again and blood to escape. It is possible, too, that a bath too prolonged and too hot may relax the vessels and permit of hemorrhage. Hemorrhage very rarely results from rupture of the cord during birth in the case of healthy children.

After the separation of the cord a slight oozing from the umbilical vessels is not uncommon. This occurs oftenest when the umbilical wound is healing slowly or has been roughly handled. Rarely the bleeding is severe.

(B) IDIOPATHIC HEMORRHAGE.—Bleeding of this variety from the umbilical wound, decidedly more common than from the vessels, is a very dangerous affection, the causes of which are diverse and little understood. Grandidier¹ writing in 1871 collected 220 cases from medical literature. Townsend² reported 14 cases in 7225 births. Winckel³ observed it in only 1 of 5000 births. The condition would appear perhaps to be more frequent than these statistics indicate, inasmuch as many cases are never reported. It occurs oftenest after the complete separation of the stump of the cord. Males exhibit it oftener than females. The general condition of the infant is of doubtful influence. It is possible that poor health of the mother during pregnancy may predispose to the development of the disease, but this, too, is doubtful. Certainly congenital syphilis appears to be a prominent cause of umbilical as of other forms of hemorrhage in the new born. Septic disease of the new born also produces it. (See p. 258.) Epstein⁴ found well-marked sepsis in 24 out of 51 fatal cases of hemorrhage. The presence of microorganisms plays an important part in the causation of many cases. Among those found in the blood or at autopsy are the streptococcus pyogenes, pneumococcus, staphylococcus pyogenes, bacillus pyocyaneus, colon bacillus, and others (Abt).⁵ Numerous cases, however, have shown no evidence whatever of sepsis. Umbilical hemorrhage is one of the symptoms of acute fatty degeneration, and especially of the hemorrhagic disease of the new born previously referred to (p. 264). The influence of hemophilia is negligible, since this disease rarely shows itself until later in life. Larrabee⁶ was able to collect but 23 cases from medical literature in which umbilical hemorrhage could be considered as dependent upon hemophilia.

Symptoms.—Severe *accidental* hemorrhage is usually sudden and profuse, and may terminate life quickly unless checked at once. The blood can often be seen flowing directly from an open vessel. It occurs generally a few minutes after birth, or, in the case of premature infants with evidences of asphyxia, during the first few hours of life, or occasionally later; sometimes even after the cord has fallen. After the separation of the cord the hemorrhage is usually in the form of oozing only. This, too, can often be seen to come from a vessel.

No general symptoms attend the accidental hemorrhage other than those of anemia, nor is there bleeding from other parts of the body.

Idiopathic hemorrhage usually occurs somewhere between the 5th and the 10th days of life, although it may occur earlier or later than this.

¹ Die freiwillige Nabelblutung d. Neugeb., 1871. Ref. Runge, Die Krankh. der ersten Lebenstagen, 1906, 224.

² Boston Med. and Surg. Journ., 1891, CXXV, 218.

³ Lehrb. d. Geburtsh., 1893, 854.

⁴ Oester. Jahrb. f. Pädiat., 1876, VII, 139.

⁵ Journ. Amer. Med. Assoc., 1903, XI, 284, for literature.

⁶ Amer. Journ. Med. Sci., 1906, March.

It is very frequently combined with the occurrence of bleeding in other parts of the body. Beginning slowly it becomes fairly free and saturates the dressing, yet it appears to be capillary in origin. Sometimes it is profuse from the start. A characteristic of the disease is that effort to check the bleeding has but a temporary effect, if any. Very occasionally the hemorrhage stops of itself for a time and then is renewed. The blood has little tendency to coagulate, but microscopic examination shows nothing abnormal in it. The infants may appear at first perfectly healthy in every other respect, or may have seemed not quite well before the hemorrhage began, or have looked ill and been somnolent or cyanotic or exhibited diarrhea, vomiting or decided icterus. In all cases decided evidence of general disturbance appears sooner or later, apart from the results of the loss of blood. Icterus often becomes intense; cyanosis may develop, if not earlier present; and hemorrhage from other parts of the body often appears, or in other instances may have antedated the umbilical hemorrhage. Not infrequently bleeding takes place not only from the navel, but into the subcutaneous tissues about it. Edema of the wrists and ankles is common. As the hemorrhage continues anemia becomes profound, and death occurs, sometimes preceded by coma or convulsions.

Prognosis.—The prognosis in accidental hemorrhage is favorable if the bleeding is not too profuse at the beginning and is discovered and checked at once. In idiopathic hemorrhage it is unfavorable. Statistics give from 25 to 35 per cent. of recoveries only. When the disease depends on sepsis, acute fatty degeneration, or syphilis, practically no hope of recovery exists. Death may occur in less than 24 hours and the average duration of life from the onset in fatal cases is only 2 to 3 days, and is seldom longer than 2 weeks.

Diagnosis.—The distinction is to be made only between the two varieties of umbilical hemorrhage. Careful inspection and the course of the case will generally make the matter clear.

Treatment.—Prophylactic treatment for *accidental* hemorrhage consists in the proper ligation of the umbilical cord not too close to the body (see page 168), in gentle handling of it afterward and in care in the removing of all evidences of asphyxia as soon as possible. Should hemorrhage occur a new ligature must be tied at once. If there is no room to apply this, or if there is free hemorrhage after the separation of the cord it may be necessary to apply a compress or even to push two needles through the skin, above and below the seat of bleeding, and ligate around these. If the bleeding is very slight the application of an astringent powder or the use of compresses moistened with tr. ferri chloridi or liquor ferri subsulphati may suffice. For the anemia which may have developed are to be employed free stimulation, abundant nourishment, the maintaining of the heat of the body, and later, measures to aid the enriching of the blood.

In the case of *idiopathic* hemorrhage prophylaxis consists, first, in attending carefully to the general health of the mother before the birth of the child and in giving to her the treatment indicated if there are any evidences of syphilis. Great care must be taken after the birth of the infant to guard against septic infection from any source. If hemorrhage begins styptic applications may be tried, such as alum or tannic acid, and especially the use of firm compresses moistened with tincture of the chloride of iron. These may be fastened in place with broad bands of adhesive plaster crossing over the navel and drawn firmly,


finishing on the back. It has also been recommended to pour moistened plaster of Paris on the navel and allow it to remain for several days. Acupuncture with the application of a ligature about the needles may be employed in the manner already described.

The administration of a 5 per cent. solution of gelatine freely by enema or by the mouth is worthy of trial. Still better is its subcutaneous use after *very carefully repeated* sterilization (p. 232).

Against the anemia the measures already mentioned are indicated.

PROTRUDING MECKEL'S DIVERTICULUM

(See also Diseases of Meckel's Diverticulum, p. 809)

 This rare affection at first sight resembles the Fungus of the Umbilicus (p. 287). It consists of a prolapse at the navel of the terminal portion of the omphalomesenteric duct which has failed to close and to disappear early in fetal life. It is generally patulous throughout, forming a fistulous tract from the umbilicus to the small intestine. In appearance it is usually a reddish tumor of glistening surface, moist with mucous secretion. Microscopical examination shows that it is of the same structure as the intestinal wall and that the surface is composed of mucous membrane. It is generally of the size of a pea or bean, although sometimes much larger, and shows a central opening through which a small amount of fecal matter may be discharged from time to time. This condition may last for years, or the duct may close spontaneously. In some cases the fistula is much larger, and the posterior wall of the intestine, and finally even quite a large portion of the bowel may project through it in the form of two intussusceptions each with its central opening.

The treatment of the protruding diverticulum, if small, consists in the application of a ligature. The larger protrusions are more difficult of cure and the prognosis is unfavorable.

CHAPTER XII

MASTITIS

The activity of the mammary glands so frequently present in the new born (see p. 60), producing enlargement with secretion of fluid, may pass into an actual inflammatory condition, and is then known as mastitis.

Etiology.—The condition is not an uncommon one, and either sex may be attacked. As a rule it occurs only in a breast in which secretion has been free and has continued for some time. Sometimes an ignorant mother or nurse is led to squeeze or rub such a breast repeatedly, with the idea that it is necessary or that the manipulation favors a good development of the gland later in life. The irritation produced, combined with want of cleanliness, favors the entrance of germs through the ducts or through fissures and suppuration follows.

Symptoms.—Mastitis begins usually in the 2d or 3d week of life, but may occur later in infancy. The breast, generally only one, grows uniformly larger, red, hot, and painful on pressure (Fig. 49). As a rule

suitable treatment employed at this period prevents the disease from advancing farther, and the inflammation subsides in a few days. In many cases, however, particularly if the squeezing of the breast is still continued, suppuration takes place and a circumscribed abscess forms. Less frequently there are multiple abscesses. Fever, restlessness, loss of appetite, sleeplessness, and loss of weight attend the suppurative process. Occasionally the inflammation spreads beyond the gland to the surrounding connective tissue, and in rare instances this perimastitis may involve much of the anterior and lateral walls of the thorax.



FIG. 49.—MASTITIS.
Male infant, aged 25 days.

Prognosis.—The prognosis of mastitis of the new born is nearly always good. Even in the cases where abscess forms recovery follows unless suppuration is very extensive. Yet permanent injury may easily remain, the secreting power of the gland in later life in females being destroyed or impaired, or the nipple being retracted or otherwise distorted.

Treatment.—The disease should be prevented by careful prophylactic measures. All pressure or rubbing of the breast of the new born is to be avoided, and cleanliness is to be enforced. If the gland is more swollen than common and inflammation is feared, a wad of aseptic absorbent cotton may be laid over it to protect from pressure and to prevent infection. If mastitis actually begins, hot, wet applications may be employed. Any abscess forming should be opened promptly and freely, and the infant given tonic treatment.

CHAPTER XIII

OPHTHALMIA NEONATORUM

Although occurring later in life as well, this form of ophthalmia is so much more prevalent in the new born that it may properly be described in connection with the diseases of this period of life. The affection is still a common one, although less so than formerly. This is particularly true of lying-in institutions, where in earlier times it often prevailed epidemically, from 5 per cent. to even 20 per cent. of the children born being affected by it. The improvement in the statistics is shown by those of

Harman¹ who found that it occurred in but 0.843 per cent. of all births in London in 1911. The greater sensitiveness of the conjunctiva in the new born certainly predisposes to the disease.

Etiology.—Infection is the cause in all instances. This is generally acquired from the vaginal discharges at the time of birth, while the head is passing through the genital canal of the mother. In other cases the infection comes from the fingers of the physician, mother, or nurse, or from infected clothing. It is also possible for the infectious matter to penetrate beneath the lids even before birth after the amniotic sac has ruptured. Although such germs as the colon bacillus, pneumococcus and others are capable of producing the disease, yet in the great majority of cases, especially if severe, the gonococcus is the cause. The milder cases may owe their origin to exposure to too bright a light, trauma, and the like, which allows a mild non-gonorrheal infection to take place.

Symptoms.—The process may affect both eyes simultaneously, or may spread from one eye to the other. Only exceptionally is but one eye involved. In the cases of a catarrhal, non-specific nature the symptoms are generally mild. They then consist of photophobia, redness of the palpebral conjunctiva, and slight serous secretion which collects in the corners or on the edges of the lids. The course is short, and the inflammation has generally disappeared in a few days.

In the gonorrheal cases the symptoms usually appear on the 3d or 4th day after birth or occasionally earlier. The first evidences are swelling and redness of the palpebral and injection of the bulbar conjunctiva, while a slight watery discharge is seen on separating the lids. The course of the disease is violent and rapid. In a few hours the lids become red, hot, stiff and extremely swollen, closing the eye tightly and being separated with difficulty by the fingers; the conjunctiva becomes much swollen with an abundant infiltration of lymphoid cells and gonococci; the secretion is somewhat more abundant and of a more purulent character; the cornea becomes involved, and in bad cases is liable to ulcerate, and perforation of it may occur even during the 2d day. In a few days the swelling of the lids diminishes, but the redness of the conjunctiva does not lessen, and, in cases which have not been benefited by treatment, folds and roughnesses develop on it giving the appearance of granulation tissue, while the secretion becomes still more abundant and quite purulent. General symptoms are absent or slight, although considerable fever may be present in the severer cases. Recovery takes place slowly. The duration is variable, the average being 3 to 5 weeks. The severe cases run 6 to 8 weeks if not influenced by treatment.

In cases where the cornea is involved this becomes cloudy, opaque and dull in appearance, and, if ulceration occurs and is followed by perforation, the aqueous humor is discharged and the iris may prolapse. Total blindness is liable to result, or the whole eye may be destroyed by a panophthalmitis. If the ulcer does not perforate healing gradually takes place and a localized opacity develops which gradually becomes more transparent. Where treatment has been successful the severity and duration of the inflammatory process are materially lessened.

Complications.—Chief among the complications is the occasional development of gonorrheal arthritis. This has been recorded repeatedly. Secondary gonorrheal infection of the nose or mouth has been occasionally recorded.

¹ Brit. Med. Journ., 1912, I, 1039.

Prognosis.—The prognosis of the severe form of inflammation is extremely grave as regards loss of vision in cases which have not received prompt and efficient treatment. Formerly a large number of cases of blindness were due to this disease. The statistics of Reinhardt¹ concerning the inmates of the blind asylums of Germany, Austria, Denmark and Holland combined covering the years 1865–1875, showed that 40.25 per cent. had lost their sight through ophthalmia neonatorum. Prompt and thorough treatment reduces the danger greatly, especially in the new born. If involvement of the cornea has already commenced before treatment is begun the prognosis is graver.

Diagnosis.—The diagnosis of the gonorrheal form is based upon the rapid and great swelling of the lids and other evidences of very severe inflammation of the eye, and later upon the free secretion of pus and the discovery of the gonococcus. Diphtheritic conjunctivitis occurs very rarely in infants and can be distinguished by the presence of a false membrane and of the characteristic bacilli, as well as by the lesser degree of swelling and the absence of the granulation-like appearance of the lining of the lids.

Treatment.—Chief in the line of treatment is careful prophylaxis. The frequency of the disease in institutions has been decidedly lessened since Credé² urged the dropping of a 2 per cent. solution of nitrate of silver into the eyes of every child immediately after birth. Of 1160 infants treated in this manner in the Leipzig Obstetrical Clinic only 1 or possibly 2 developed ophthalmia; *i.e.* 0.086 to 0.172 per cent.; whereas earlier statistics based on 4057 infants in the same maternity had given 7.8 per cent. suffering from ophthalmia. Observations of most later investigators are in entire accord with the results obtained by Credé. The use of an antiseptic vaginal injection before labor is also to be recommended. In private practice the instillation and the vaginal douching should be employed if there exists any probability that the vaginal secretion of the mother may be gonorrheal in nature. Newer silver compounds have been recommended to replace the nitrate solution. Thus a solution of protargol (5 to 20 per cent.) or of argyrol (25 per cent.) has been employed with success instead of that of nitrate of silver, but would appear to be less certain in its results. The water in which the child is bathed must never be used for the washing of the eyes. In cases where the danger of gonorrheal infection is entirely unlikely the eyes should be thoroughly cleansed after birth with cotton moistened with distilled water or a saturated solution of boric acid. To prevent the milder form of conjunctival inflammation the eyes of the new-born infant must be protected from too bright a light and from mechanical injury.

When gonorrheal ophthalmia develops in an infant in a maternity or a hospital ward the patient should at once be isolated, and the nurse in charge should have nothing to do with the other children. In all cases of the disease the cloths, cotton, and the like used about the eyes should be promptly destroyed. The sound eye, if one only is affected, must be carefully protected by covering it with a wad of antiseptic cotton and enveloping with a gauze bandage. The hands of the child must also in many cases be restrained by bandaging them to the sides or in other ways. The two chief indications in treatment are the repeated thorough

¹ Zweiter europ. Blindenlehrer-Congr., Dresden, 1876. Ref. Magnus; *Die Blindheit*, 1883, 165.

² Arch. f. Gyn., 1881, XVII, 50; 1883, XXI, 179. *Die Verhütung d. Augenentzündung d. Neugeborenen*, 1884.

disinfection of the eye, and the reduction of inflammation by cold applications. In the mild cases, not of gonorrheal nature, all that is required is to wash the eye thoroughly several times a day with a saturated solution of boric acid dropped into it from a blunt-pointed medicine dropper. In the gonorrheal cases the treatment must be more energetic. The lids should be separated, and the boric acid solution instilled every $\frac{1}{2}$ to 1 hour. The solution should be dropped in the corner nearest the nose with the child's head turned a little toward the diseased side so that the fluid may run into the eye, and then out as far removed as possible from the unaffected eye. Care must be taken that the fluid penetrates beneath the upper as well as the lower lid. Thorough frequent removal of pus must be accomplished in this way, since the retention of the secretion is very harmful. A solution of corrosive sublimate of a strength of 1:10,000 may be used instead of boric acid. In addition to the washing there should be instilled into the eye once a day 2 or 3 drops of a 1 per cent. solution of nitrate of silver; or a 10 to 25 per cent. solution of protargol or argyrol more frequently. To reduce the inflammation and swelling of the eye cold compresses must be applied. These are best made of little squares composed of 1 or 2 layers of soft linen cloth cooled on a block of ice, and changed every 2 or 3 minutes for 30 minutes or more, with intermissions of 2 or 3 hours. In very bad cases the application must be kept up continuously until the swelling has abated. Gonorrheal ophthalmia is, however, such a serious affection that whenever possible the treatment is more safely entrusted to an ophthalmologist.

CHAPTER XIV

SCLEREMA AND EDEMA

SCLEREMA NEONATORUM

(Sclerosis; Scleroma Sclerema; adiposum)

Probably several different conditions were formerly considered to be identical with that which we now designate as sclerema. The affection is sometimes wrongly called scleroderma, which is at present believed to be entirely distinct from this. Sclerema neonatorum in its modern sense appears first to have been described by Uzembezius.¹

Etiology and Pathology.—Although not a common disease anywhere, it is seen much more frequently in Europe than in America. Writing in 1897,² I was able to discover but 5 undoubted and fully detailed cases published in the United States, and to these I added a 6th, and later reported another.³ It is most often seen in premature or in greatly debilitated new-born infants, living under very unfavorable hygienic conditions, and it has occurred oftenest in institutions for foundlings. At a slightly later period, in infants a few weeks or months old, it occasionally develops following a severe diarrheal affection or other exhausting disease. The nature of the change which takes place is undetermined. An explanation with much in its favor is that the great

¹ *Ephemerides naturæ curiosorum*, 1722. Ref. Hennig, Gerhardt's *Handb. d. Kinderkr.*, 1877, II.

² *Medical News*, 1897, Oct. 2.

³ *Arch. of Ped.*, 1906, Feb.

lowering of the body temperature dependent upon the condition of inanition present produces a hardening of the subcutaneous adipose tissue (Knöpfelmacher).¹ That this can occur might be due to the low percentage of olein and the high percentage of stearin and palmitin in the fat of infants, and the consequently higher melting point as compared with the fat of adults. Yet it is to be noted that the great majority of new-born infants who develop collapse-temperatures do not suffer from sclerema. Various microorganisms have been considered to be the active causative agents, but the existence of any such influence is very uncertain. The etiology is still undetermined.

Pathological Anatomy.—There are no characteristic lesions. Incision of the skin is not followed by any exudation of blood or serum as in edema neonatorum. The subcutaneous connective and fatty tissues appear unusually dry. Microscopical examination showed nothing whatever abnormal in a case reported by Northrup,² while Ballentyne³ discovered a growth of bands of connective tissue and an atrophy of the fat cells, and the connective-tissue hypertrophy was confirmed by Sarbonat.⁴

Symptoms.—The two characteristic symptoms are fall of temperature and a hard swelling of a portion of the skin. Occasionally at birth, but oftener when the greatly debilitated child is a few days old, induration of the skin is discovered. This change begins usually in the feet and calves and rapidly spreads perhaps over the whole body. It is usually most decided in the cheeks, buttocks, back and thighs. The swollen tissues seem to have an almost stony coldness and hardness and will not pit on pressure. The limbs, and sometimes the whole body, are more or less stiff and immovable. The skin cannot be lifted from the subcutaneous tissue. It is pale, waxy, and sometimes in places discolored bluish or yellowish, resembling an old bruise. The temperature of the body is generally very low, sometimes not over 90°F. (32.2°C.) in the axilla, or even less than this. The child has an almost inaudible cry. The respiration and cardiac action are very feeble; the fontanelle is sunken; the infant becomes somnolent and will not take nourishment. Exceptionally the disease is complicated by edema (Parrot).⁵ Atelectasis is very prone to develop.

Occasionally cases are not so severe, the temperature is not so low, nor does the child show so great a degree of inanition and debility.

Prognosis.—This is extremely unfavorable. The great majority of cases die in a few days. Occasionally when the disease is not too extensive, the general condition of the infant is better, and treatment commenced early, recovery will slowly take place. I have observed at least 3 such recoveries in cases which seemed to be properly designated sclerema.

Diagnosis.—Sclerema is to be distinguished by the adherence of the skin to the subcutaneous tissues and the fact that it cannot be pitted, the very low temperature, and the marked rigidity of the body. The first two symptoms serve to distinguish it from edema. Scleroderma is generally considered a disease of adults, but it is possible that some of the milder cases called sclerema may properly be placed in this

¹ Jahrbuch für Kinderheilkunde, 1897, XLV, 177.

² Transactions American Pediatric Society, 1889.

³ Antenatal Pathology and Hygiene, 1902, I, 75.

⁴ Arch. de méd. des enf., 1906, IX, 22.

⁵ La clinique des nouveau-nés, 1877, 127.

category. In scleroderma the process of hardening is more local, the course is chronic and there are no general nutritional symptoms or fall of temperature. (See studies by Cockayne¹ and by Mayerhofer.²)

Treatment.—The only treatment possible is the maintenance of the bodily temperature, the employment of general massage with oil, and the sustaining of life by food and stimulants. The continuous application of hot water bags to the affected regions have been of benefit in some of the milder cases, and Wolff³ has had good results in severer cases by submitting the infant to a continued temperature of 40° to 42°C. (104° to 107.6°F.). The original cause, if discovered, and complications, especially atelectasis, must receive treatment appropriate for them.

EDEMA NEONATORUM

(Scleredema. Acute Edema)

Although edema from various causes may develop in infancy, there is a form oftenest seen in the new born to which the name of Edema Neonatorum is given. This condition is considered by some writers to be an edematous form of sclerema.

Etiology.—The disease is uncommon. In some cases septic infection appears perhaps to have been responsible and in others the existence of a specific infectious agent has been suspected. Exposure to cold has also been considered to be sometimes the cause. It is more likely to develop in weakly or premature children, in foundling asylums, or under bad hygienic conditions, but hearty infants are exceptionally attacked. Disturbance of the respiratory and circulatory functions and beginning nephritis have each been considered the causative agent.

There has further been described by Schridde⁴ and the observation confirmed by others (Chiari;⁵ Wienskowitz⁶) a special form of widespread edema which is congenital and which has been designated by Chiari "fetal erythroblastosis," characterized by a universal anasarca; hydrops of the serous cavities; enlargement of the liver and spleen; a very large number of erythroblasts in the blood, with a great diminution of other elements; numerous erythroblasts in the liver, spleen, kidneys and other organs; and an abnormal deposit of hemosiderin in the spleen and liver. This form of edema is seen chiefly in premature infants born of mothers with nephritis, which has been the cause probably of a toxic disturbance of the blood-making functions.

Pathological Anatomy.—The skin is hard as in sclerema, but incision into it after death allows a serous fluid which is colorless, or of a slightly bloody tint exuding from the subcutaneous tissue. The other findings in the skin are uncharacteristic and vary with the case. The lesions of the form described by Schridde have already been mentioned. The alterations in other parts of the body are chiefly those of atelectasis and inanition.

Symptoms.—In the congenital cases a universal edema is seen at birth. In other cases the disease usually begins in the first 3 or 4 days of life, although sometimes not until the infant is some weeks or even

¹ Brit. Journ. Child. Dis., 1916, XIII, 225.

² Jahrb. f. Kinderh., 1915, LXXXI, 348.

³ Monatsschr. f. Kinderh. Ref., 1914, XIV, 66.

⁴ Münch. med. Wochenschr., 1910, LVII, 397.

⁵ Jahrb. f. Kinderh., 1914, LXXX, 561.

⁶ Berl. klin. Wochenschr., 1914, LI, 1725.

1 or 2 years old. It is ushered in, as a rule, by swelling of the calves, which rapidly spreads over the lower extremities and in the course of at most 2 or 3 days over the rest of the body. The skin is pale, shining and tense. Pressure gives a doughy sensation and produces pitting. The skin is not immovable over the underlying tissues as in sclerema. The limbs are very cold, stiff and difficult to move; the face is stiff and expressionless. The infant is in a condition of collapse, with feeble pulse and respiration, feeble cry, inability to suck, very low bodily temperature, and diminished secretion of urine. At first there is restlessness, but finally a soporous state. In fatal cases the condition gradually grows worse with rapid wasting, and the child dies after a few days, or at most 1 or 2 weeks. The disease may be complicated by pneumonia, atelectasis or septic conditions. It may also occur in connection with sclerema. Icterus and gastrointestinal disturbances may be present.

Prognosis.—The prognosis of edema neonatorum is, as a rule, bad. Cases recover but rarely.

Diagnosis.—The disorder resembles sclerema in many particulars. It is distinguished from it, however, by the ability to pit the skin and to move it over the underlying tissues. It is to be remembered also that an edema is seen in erysipelas and in acute nephritis in the new born, and is not uncommon in advancing marantic states in many infants. In the latter condition, however, the dropsy comes on more slowly, and is seen oftenest about the ankles, hands and scrotum, although sometimes in the face as well. The temperature, too, is usually not so low, and the cause of the edema is generally evident.

Treatment.—The condition can better be prevented than cured. Feeble children must be kept warm, and nourished in the best manner possible, and all depressing diseases treated energetically. If edema has already developed these same methods are still to be employed. Alcoholic stimulation and remedies to sustain the heart's action are necessary, together with gentle massage of the body with oil. Complications require treatment appropriate to them.

CHAPTER XV

TRANSITORY FEVER IN THE NEW BORN

(Inanition-Fever; Hunger-Fever; Thirst-Fever, etc.)

The natural irregularity of the temperature in the early weeks of life readily predisposes to a febrile elevation from very slight causes. Temporary rises of temperature are probably much more frequent than is usually supposed; the mistake being the result of the common absence of any systematic temperature-taking in infants which appear to be in health. Thus Lo Cicero¹ by making observations on 83 infants every 3 hours during the first 8 days of life found that in 49 there were sudden rises of temperature to from 37.5° C. to 39°C. (99.5° to 102.2°F.), with sudden falls, without any objective signs except somnolence or sometimes restlessness. Eross² noted elevation of temperature in 431 out of 956 new-born infants, viz.; 45.08 per cent.

¹ *La Pediatra*, 1915, XXIII, 768.

² *Jahrb. f. Kinderh.*, 1891, XXXII, 68.

The fever usually appears, from the 2d to the 4th day of life. The elevation may last but a few hours or continue for several days. (Fig. 50). The general condition of the infant seems to be but little influenced, children strong and of good weight at birth being affected as readily as others. The **causes** are not clearly understood. That the fever is of so transitory a nature and without discoverable lesions largely eliminates such factors as general sepsis, pneumonia, and other grave inflammatory conditions; although Eröss believed that in many instances the elevation depends upon a slight temporary infection from local putrefactive changes in the umbilical cord, and in others upon a disturbance of the digestive tract. This explanation doubtless applies to a number of cases; but the

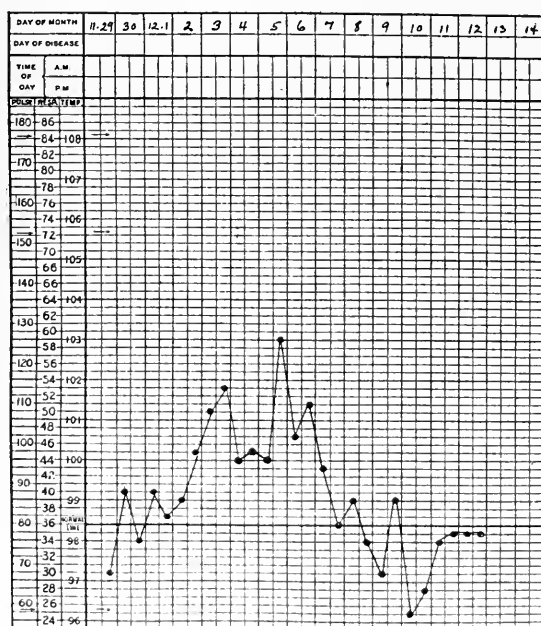


FIG. 50.—TRANSITORY FEVER IN THE NEWBORN.

Baby M., born Nov. 29. Persistent fever and loss of weight. Weighing before and after each nursing showed an insufficient amount of milk furnished. After supplemental feeding was commenced Dec. 6 the temperature fell and gain in weight began.

rapid development of the rise of temperature and its rapid disappearance, as well as the uniform occurrence at the same period of life, makes it unlikely that the majority of cases can be attributed to such causes. It has also been maintained that the fever depends upon irritation of the kidneys by uric acid present in them, but there is lacking sufficient evidence in support of this view. The fact that the fever is liable to develop at the time when the initial loss of weight is at its greatest is a suggestion that it may be associated with a condition of inanition from hunger (Holt)¹ or may depend upon thirst with consequent desiccation of the tissues (Müller).² Those infants who lose most in weight are the ones oftenest showing elevation of temperature. It has been found, too, that

¹ Arch. of Ped., 1895, XII, 561.

² Berl. klin. Wochenschr., 1910, XLVII, 673.

the giving of breast-milk freely, or even of water, will often cause the fever to disappear promptly. There seems every reason to believe that these causes are operative in many instances; but against the view that they are the sole cause is the fact that in hunger-states after the period from the 2d to the 4th day, the starving infant with drying tissues is more liable to exhibit subnormal bodily temperature. The fact that the fever appears, as stated, only upon certain days of life, and that it does not attack with greater frequency those already feeble or of low temperature at birth, would support the suggestion which has been made that it may depend upon an autointoxication, the result of some of the physiological processes which are especially active at this time, and that the giving of water or breast-milk freely may act not directly by relieving hunger or thirst and consequent inanition, but by favoring a washing out of these toxic products from the system.

The **symptoms** attending transitory fever are more or less characteristic. The appetite is often diminished, and the children nurse poorly from the breast or the bottle. In other cases they suck with avidity and an evidence of hunger. There is restlessness or in more severe cases prostration and little movement. Loss of weight continues while the fever lasts, and is usually greater than in normal infants. The temperature reaches 39°C. (102.2°F.) or often more. Its duration is 2 to 3 days, sometimes with intermissions, and the fall is generally rapid.

The **prognosis** is good in the majority of cases, except that the continued and increasing loss of weight may exert a serious influence upon the infant, and even predispose to a fatal termination if any other affection arises. The **diagnosis** is on this account very important. A careful examination will generally reveal the causes of fever of any other nature than that now under consideration, and such an examination must, of course, invariably be made. Septic inflammation, if other than purely local, is generally excluded by the disposition to develop at a slightly later period; although this is by no means an invariable rule. The fever also is not of such short duration and the infant seems more ill. The **treatment** in all cases dependent upon insufficient food or liquid is simple. In addition to the free supply of breast-milk or of water, other measures are entirely symptomatic.

SECTION III

THE INFECTIOUS DISEASES

CHAPTER I

GENERAL

DEFINITION

Much confusion has existed regarding the employment of the terms "infectious" and "contagious" as applied to disease. In the widest sense an "infectious" disease is one due to a specific living organism of some sort which "infects" the human body; while "contagious" indicates merely that the disease may be contracted by one individual by contact, directly or indirectly, with another. As a matter of fact most of the infectious diseases are contagious also. A notable exception to this is seen in the case of malaria. Certain diseases, such as some of those dependent upon the presence of various parasites in the gastro-enteric tract, the contagious parasitic affections of the skin, and certain others, while clearly infectious in the broader sense are not ordinarily classified among the acute infectious disorders; that term, in the narrower sense, and for the sake of convenience, being reserved for those dependent upon a more general infection of the human organism by microscopic germs, in which the symptoms are of a more general nature. Those with a comparatively short and self-limited course are called the "acute infectious diseases;" or, if febrile, the "acute infectious fevers." The others are designated "chronic infectious diseases". Many of the first class are attended by special cutaneous eruptions, and to those the title "acute exanthemata" is often applied. The course of the infectious diseases is often marked by certain stages or periods designated by special terms. That during which the germs are developing in the body without characteristic manifestations is called the *period of incubation*. On this follows the *prodromal stage* or *period of invasion*, during which the *initial symptoms* appear. After this is the stage of the fully developed disease, or *stage of florition*, in the case of the exanthemata called the *period of eruption*. Last comes the *stage of decline*, in the eruptive fevers sometimes designated the *period of desquamation*.

Any accurate classification or even enumeration of the infectious disorders is impossible at this time of rapidly advancing knowledge regarding them. The list is constantly growing. Among them are included here: scarlatina; rubeola; rubella; variola; vaccinia; varicella; typhoid fever; erysipelas; cerebrospinal fever; diphtheria; grippe; pertussis; mumps; malaria; tetanus; and poliomyelitis. The chronic infectious diseases include tuberculosis and syphilis.

Pneumonia appears to be quite certainly an infectious disorder, rheumatism and perhaps chorea probably so as well; but for the sake of convenience and for other reasons I have considered them elsewhere. So, too, certain intestinal infections, such as cholera and some forms of enterocolitis, are clearly infectious and communicable; yet they are more

conveniently studied in the section on Diseases of the Intestine. The septic infections of the new born have for the most part already been described in a preceding section, and gonorrhea is discussed under the headings of the organs affected.

At times two or even more acute infectious diseases may affect a patient simultaneously or one shortly after the other. The following of pertussis upon rubeola is of great frequency, and the development of diphtheria with or immediately after scarlet fever or especially measles is also often seen. Measles and scarlet fever may occasionally occur together, and even epidemics of this have been reported. The combination of typhoid fever with scarlet fever is less often reported, and that of typhoid fever with measles still less frequently. In one instance I saw rubeola and diphtheria with varicella which took on a gangrenous form occur nearly simultaneously, and in another typhoid fever, measles and cerebrospinal fever.

METHOD OF DISSEMINATION

The older views were long accepted without question that the chief means of the dissemination of the germs of infectious diseases was by the air-current, the dust of the room, the playthings, books and clothing of the patient, and the clothing of attendants or other third persons. More careful study of recent years has proven with fair satisfaction that, although these methods are theoretically possible, in practice they occupy a very limited position. The transmission by a third person is practically only by the direct handling of a child, as by a nurse who has just left an infectious patient close by. This could take place only with great carelessness and in hospital wards. The chief source of infection is close contact with or proximity to the patient himself, the disease being communicated to others either before it has been recognized and the individual isolated, or given after quarantine has been removed and the patient believed to be well. It is usually by means of the mucous secretion of the infected person; often through the microscopic drops of mucus which are coughed or sneezed out by the patient or carried upon the breath; these reaching the respiratory tract of the previously healthy individual. In numerous instances the disorder has been perhaps so mild that it has not been recognized at all, and the child has remained an undiscovered source of infection. Those who have recovered from an infectious disease, but who still harbor its active infectious germs are called "carriers," and the same title is applied to the smaller, but still quite large class comprising those who have not suffered, but who have been in close contact with patients, and who carry the germs upon their mucous membranes. It is almost solely in this way that the disease is transmitted by a third healthy person, and the danger is probably less than from those who have suffered from the disease. The possibility of the dissemination by dust, water, insects, and in other ways will be considered under the headings of the various infectious diseases. It varies with the disease; tuberculosis, for instance, being undoubtedly readily spread by dust upon the hands or in the food; typhoid fever by water and by insects.

GENERAL RULES FOR THE HYGIENIC CARE AND THE QUARANTINE OF INFECTIOUS DISEASES

The diseases under consideration vary greatly in their relative infectiousness, and in the mode by which the germs are chiefly conveyed. The following rules are those to be applied where rigorous prophylactic

measures are to be carried out. Circumstances may alter either the possibility or the necessity of their application. It must be understood clearly that the methods to be described constitute in some respects the ultimate limit of precautions to be followed; not those which are considered by the leading authorities as actually necessary. It has, for instance, repeatedly been shown in hospital practice that it is perfectly possible, with proper precaution by the aid of the "box method," to treat different infectious diseases in the general wards without extension taking place. (See Koplik,¹ von Pirquet,² Richardson,³ and Rundle and Burton⁴ on the Box System and similar methods in hospital practice.) However, inasmuch as the requirements of local Boards of Health vary and are often rigorous, and since our knowledge of the method of spread of infectious diseases is still incomplete, it is better to err upon the safe side, and the full details of quarantine may well be described.

Room.—The room selected should be preferably in the upper portion of the house. Here the patient is less liable to be disturbed by noises, and here, too, isolation can be better carried out. It should be well-lighted and well-warmed, and capable of being well-ventilated. It should open into another room, which we may call the anteroom. This should be, if possible, a bath-room or a room opening into it. The temperature of the sick-room should be from 65 to 70°F. (18.3° to 21.1°C.). The door entering it from the hallway should be locked, and the cracks stuffed with paper or cotton. The door from the anteroom into the hallway should be kept closed except at the time of exit or entry. A sheet should be hung here which should be kept moistened with a solution of corrosive sublimate (1:1000) or one of carbolic acid (5 per cent.). The real value of this measure against the spread of the disease is more than questionable, but it serves at least as a reminder, and satisfies the demands of some Boards of Health.

Ventilation is best obtained by a fireplace, or, in the absence of this, from the anteroom. Screens should be used at the windows of the sick-room to prevent the direct action of draughts upon the child and the entrance of insects. If the weather permits, the window of the anteroom should be opened constantly, thus practically cutting off the sick-room from the rest of the house. It is to be understood that the action of draughts in producing chilling of the surface and any consequent damage is not so much to be feared during the febrile state of the disease. It is after fever is past that injury may follow.

The sick-room should have all unnecessary articles removed, especially all clothing, carpets, curtains, upholstered furniture, and pictures. Only such books and toys may remain as shall afterward be destroyed. The room should be kept very clean, all dust being removed by wiping with a 1:1000 bichloride solution. The solution must not be used upon metal.

Patient.—The food for the patient and nurse should be brought to the door of the anteroom by an attendant. After the dishes, spoons or other articles have been used, they should be washed, and either boiled, or disinfected with a 5 per cent. solution of carbolic acid in which they should lie for some time. All bed-clothes and body-clothing should be changed frequently and should be immersed in a 5 per cent. solution of

¹ Arch. of Ped., 1911, XXVIII, 728; 1912, XXIX, 5.

² Zeitschr. f. Kinderh., Orig., 1913, V, 213.

³ Jour. Amer. Med. Assoc., 1913, LXI, 1882.

⁴ Lancet, 1912, I, 720.

carbolic acid or a 1:1000 solution of corrosive sublimate kept in the anteroom. After thorough soaking they should be wrung out, placed in a bucket and taken by an attendant from the nurse at the door of the anteroom. They may now be washed with the other linen of the house without danger. Old linen or muslin cloths and absorbent cotton may well replace handkerchiefs, since they can be destroyed at once after use. The utmost care should be taken to receive and disinfect or destroy promptly all discharges from the eyes, nose, mouth and, in the case of typhoid fever, the intestine and bladder. When there is free expectoration, paper sputum-cups or others holding a 5 per cent. solution of carbolic acid may be employed.

Attendants.—The nurse should be dressed in washable material, and should have a cap to cover thoroughly the hair and the neck. She should have her meals in the sick-room or, better, anteroom, and leave it only when about to pass through the house on her way out. Before doing this she should slip off her outer garment, disinfect her hands and face with a 2 per cent. solution of carbolic acid or a 1 : 5000 of corrosive sublimate, put on her outer street clothes, which should be kept in the anteroom, and go directly out without stopping to talk with members of the family. Those of the family who must necessarily enter the room to relieve the nurse should adopt similar precautions. With proper care the confinement of the nurse to the sick-room during the whole of the disease is entirely unnecessary and constitutes a superfluous hardship. The physician too, should on entering the anteroom array himself in a linen or rubber garment. A linen dust-coat is very serviceable for this purpose. He should wear an oil-silk or other cap upon his head. Before leaving the anteroom he should remove these articles, and disinfect his hands and face carefully.

Family.—Other non-immune children should, when possible, be removed from the house, and should not be allowed to go to school or mingle with others until a time has elapsed equal to the duration of incubation, for fear they may have the disease already in their systems. If they are obliged to continue to reside in the house they should not attend school or associate with other children until the patient is removed from quarantine. In case, however, they have already had the disease, there is no actual reason why they may not safely be with other children if the measures for isolation of the patient are being properly carried out. The feeling of other parents regarding the matter is, however, so strong that it is useless to insist upon the truth of this.

Final Disinfection.—When the disease is over the child should receive a disinfecting bath of corrosive sublimate 1 : 10,000, particular attention being paid to the hair. (See p. 243.) He should then be dressed in entirely clean underclothing and removed to another room. The outer clothing in use at the time the disease began should have been meanwhile thoroughly exposed to fresh air and sunlight. The sick-room and all others used during the illness should be thoroughly disinfected. The carpets, if they have not been removed previously, and the mattresses and pillows should be subjected to superheated steam in a municipal disinfecting plant, when this is practicable. When not, they may be allowed to stay in the room during fumigation, and then exposed to the open air and sunlight for several days. The walls, if papered, should be scraped, and, if painted, washed with a 1:1000 solution of corrosive sublimate. The floor should be scrubbed with soap, and then washed with the solution. The furniture should be wiped carefully with the solution, except-

ing in the case of metal, when carbolic acid, 5 per cent., must be used instead. All small objects, such as toys, books and the like should be burned. Fumigation with formaldehyde has been much in vogue, but is being with reason abandoned in many quarters. As ordinarily performed it is questionable whether it possesses any value. It may be regarded as an additional precaution. Preparatory to the fumigation the cracks of the windows should be plugged with cotton, a formaldehyde lamp or candle started or the solution scattered about, the outer door then closed and the cracks plugged in the same way, and the room left undisturbed for 12 hours.

CHAPTER II

SCARLET FEVER

(Scarlatina)

History.—Although by some believed to have been known to the ancients, or at least to early Christian or Arabian writers, it is more probable that scarlet fever was not distinguished from other eruptive fevers until about the middle of the 17th century. At this time Sennert¹ gave a good clinical description of an epidemic. Sydenham² appears to have been the first to distinguish it clearly from measles, and to apply to it its present name. The disease has certainly increased greatly in frequency since the 17th century, and now is one of the most common of the infectious fevers, without any discoverable diminution in its incidence (Donnelly).³

Etiology. Predisposing Causes.—Climatic and geographical conditions seem to possess but little influence. The disease appears capable of developing wherever the infection is brought, although it still remains far most common in Europe and America. The season of the year is perhaps not without importance, the greatest number of cases occurring in the winter and especially in the autumn months. This is, however, a much disputed point, and the influence of season, if it exists, is certainly not very considerable.

Sex, race, social conditions, and sanitary surroundings possess no certain predisposing causal relationship, the statements regarding them being entirely contradictory. So, too, the previous state of health of the patient has no discoverable connection with the susceptibility, except that patients with wounds seem particularly disposed to contract the disease. (See Surgical Scarlet Fever, p. 316.)

On the other hand, the influence of *age* is very great. Although the disease may possibly occur congenitally (Gigon),⁴ or may develop in old age, by far the most frequent occurrence is in childhood. The greatest number of cases were found by Gresswell⁵ between the ages of 5 and 10 years, and the next between 2 and 5 years. Donnelly⁶ in a review based upon some millions of cases placed about 50 per cent. between 3 and 8 years, and 90 per cent. in the first 15 years of life. Under the age of 1 year, and espe-

¹ Med. pract. Wittenberg, 1651, II, cap. 12. Ref. Billiet and Barthéz (Sanné) *Mal. des enf.*, 1891, III, 74.

² *Processus Integri*. Ref. Williams, 20th Cent. Pract. of Med., 1898, XIV, 117.

³ *Amer. Jour. Dis. Child.*, 1916, XII, 205.

⁴ *Jahrb. f. Kinderh.*, 1910, LXXII, 676.

⁵ A Contrib. to the Nat. Hist. of Scarlatina, 1890.

Loc. cit.

cially under 6 months it is very much less common than at other periods of early life. Gresswell gives only 0.6 per cent. in the 1st year in 588 cases; Caiger and Dudgeon¹ 0.9 per cent. in 167,840 cases of the Metropolitan Asylums Board's Hospitals, and Herberg² 0.6 per cent. in 1000 cases. Lemarquand³ records 22 personal observations, and gives a number of others from medical literature, in which nursing women with scarlet fever did not communicate the disease to their infants in the early months of life. The youngest patient under my own observation was 3 months of age.

The *individual susceptibility* to the disease varies greatly. Some individuals appear immune, although repeatedly exposed. The disease certainly has a less general tendency to spread than some of the other infectious disorders possess. In the experience in the Faroe Islands (Hoff),⁴ for instance, while 99 per cent. of the population of one of the chief towns showed susceptibility to measles, only 38.3 per cent. of unprotected persons contracted scarlatina. In Boston during 5 years the number of cases varied from 16.77 to 62.87 per 10,000 of the population (McCollum).⁵ My own experience has always shown the spread of scarlatina to be much more readily controllable than that of measles.

The comparatively small number of cases occurring in early infancy and after childhood is passed is probably in part due, in the first instance, to a lesser frequency of exposure, and, in the second, to the protection already given by a previous attack. Still, a greater degree of susceptibility certainly does appear to exist in childhood, since in the Faroe's epidemic, where no previous immunity existed, 56.3 per cent. of the cases were under 20 years of age and only 7.6 over 40 years.

An *epidemic influence* is also discoverable. This is especially seen in country districts, for in many of the larger cities the disease is practically endemic, cases constantly appearing although in very different numbers at different seasons and in different years. The severity of the disease and the consequent mortality vary greatly with the epidemic.

Of great scientific interest is the much disputed possibility of the *experimental transmission* of scarlet fever to apes, particularly as bearing upon the possible etiological influence of the streptococcus. While successful transmission has been reported by Landsteiner, Levaditi and Prasek;⁶ Bernhardt,⁷ and Schleissner,⁸ this has not been confirmed by the studies of Draper and Hanford;⁹ Krumweide, Nicoll and Pratt;¹⁰ Klimenko,¹¹ and others. It must be concluded that the possibility of transmission to animals still lacks definite proof.

Exciting Cause.—The direct cause of the disease is infection, and analogy is convincing that this is by means of some form of living organism. The *nature of the germ* has been much discussed, but still remains disputed. From the time of the report by Klein¹² of the streptococcus scarlatinae to the present, various germs have been described.

¹ Allbutt and Rolleston's Syst. of Med., 1906, II, 1, 429.

² Zeitsch. f. Heilk. u. Infektionskrankh., 1910, LXV, 237.

³ Thèse de Paris, 1906, July.

⁴ Sundhedskolligiets Aarsberetning 1876. Ref. von Jürgensen in Nothnagel's Encyclopedia, Amer. Edit., Article Measles 228; Scarlet fever 382.

⁵ Boston City Hospital Reports, 1899.

⁶ Ann. de l'Institut. Pasteur, 1911, XXV, 754.

⁷ Deut. med. Wochenschr., 1911, XXXVII, 791.

⁸ Jahrb. f. Kinderh., 1915, LXXXII, 225.

⁹ Jour. Exper. Med., 1913, XVII, 517.

¹⁰ Arch. Int. Med., 1914, XIII, 909.

¹¹ Jahrb. f. Kinderh., 1913, LXXXVII, 679.

¹² Proc. Royal Soc., London, 1887, XLII, 158.

Among the different investigators Class¹ discovered a diplococcus which he believed to be pathogenic, and Baginski and Sommerfeld² reported a similar germ as being uniformly present in the pharynx and as found in the blood and organs of fatal cases. Salge³ and others have produced agglutination of the scarlatinal streptococci by the blood from a case of scarlet fever, but Kolmer⁴ found agglutinins and antibodies present in only a small percentage of cases. Mallory⁵ reported a protozoan-like body, the *cyclastis scarlatinalis*, resembling the malarial parasite and found in and between the epithelial cells of the skin and in the lymph vessels and spaces of the corium; and in a later publication with Medlar⁶ described a Gram-positive bacillus constantly present early in the case in the crypts of the tonsils. Later these disappear and are replaced by streptococci.

The chief point of interest is connected with the possible causative influence of the streptococcus. There is reason to believe, as pointed out by Hektoen,⁷ Jochmann,⁸ and others that, although a germ of this class is frequently present, it is rather the cause of complicating conditions than of the disease itself. The real nature of the causative organism of scarlet fever appears to be still unknown.

Method of Transmission.—The disease never originates spontaneously, but is always transmitted from the sick to the well. The transmission is either direct, and this is true of the great majority of cases, or mediately through clothing, carpets, bedding, and the like, or a third person. The mediate infection is certainly uncommon. (See *Infectious Diseases*, p. 306.) The transmission occurs with great frequency in schools, where children with the disease about to manifest itself, and who consequently possess the power of infecting, communicate it directly to others.

Numerous instances are on record where the disease has been carried even for some time and distance in the clothing of physicians or nurses, or been transmitted by letters, toys and books, or carried by animals. These methods of dissemination are, however, exceptional. There are many instances of its spread by milk coming from dairies where scarlet fever existed among the families of the employees. Kober⁹ found the disease at the dairy or milk-farm in 68 out of 99 reported epidemics of scarlet fever. Although instances are reported, it is yet doubtful whether an immune inmate of a house where scarlet fever exists, who does not come into intimate association with the patient, can transmit it to another individual. Indeed, close contact with the patient or with the infected article seems to be necessary, as the germs do not appear to be carried to any distance by the air. The disease has also been communicated by direct inoculation (Stickler).¹⁰

Seat of the Virus.—The parts of the body in which the infectious matter is chiefly situated is a subject still incompletely determined. The fresh or dried mucus from the nose and throat is undoubtedly infectious,

¹ New York Med. Record, 1899, LV1, 320.

² Berl. klin. Woch., 1900, XXXVII, 588.

³ Münch. med. Woch., 1902, Oct. 14.

⁴ Arch. Int. Med., 1912, IX, 220.

⁵ Journ. Med. Research, 1904, X, 483.

⁶ Journ. Med. Research, 1916, XXXIV, 127.

⁷ Journ. Amer. Med. Assoc., 1903, XI, 685; 1907, XLVIII, 1158.

⁸ Deut. Arch. f. klin. Med., 1908, LXXIII, 209.

⁹ Amer. Journ. Med. Sci., 1901.

¹⁰ N. Y. Med. Record, 1899, LV1, 363.

as is the pus from a complicating otitis, empyema or suppurating adenitis. The infectiousness of the urine and feces is in doubt. It is questionable whether the expired air carries the germs. The scales of the epidermis were formerly considered extremely infectious, but this is now much disputed, and there is little if any danger from them except as they may be merely the carriers of contagious material derived from the secretions, which has attached itself to them.¹ It is certain that the disease can be given by persons suffering from a scarlatinal angina who have never exhibited eruption or desquamation.

Period of Maximum Infectivity.—With this uncertainty as to the chief method of dissemination of the contagion, the question regarding the most infectious stage must remain as yet undetermined. The ease with which infection spreads in schools shows that the latter portion of the stage of incubation or certainly the earliest part of the stage of invasion must possess infectious power. Probably the most active period is that of the height of the eruption. The disease is also communicable during the stage of desquamation and after it. This is proved by the frequency with which it occurs in other children in the family, after patients who have suffered from scarlet fever in hospitals have returned to their homes ("Return cases"). Thus Pugh² found that 2.9 per cent. out of 6507 hospital cases later communicated the disease to others at home after 6 weeks in the hospital. In many of these instances the virus had probably persisted in the nasal or faucial secretion of a mucous membrane still slightly diseased, or in the pus of a discharging ear.

Tenacity of Life.—The remarkable tenacity of life of the germs renders difficult the solution of the question of the time of greatest infectiousness. There are numerous carefully observed instances on record which seemed to prove that the germs may live and communicate the disease after months or even after several years. Murchison³ gives an instance of the persistence of vitality for 4 months; Lommel⁴ for 133 days; and Sannés⁵ for 71 days. Others of still longer duration are on record but are open to doubt.

Pathological Anatomy.—There are no definite lesions characteristic of the disease except that of the skin and of the mucous membrane of the mouth and throat. The intense hyperemia present during life disappears after death. There is found, however, a dilatation of the lymphatics and blood-vessels of the *skin* (Pearce),⁶ with a swelling of the cells of the rete, sometimes with extravasations of blood between them. An infiltration of all the layers of the skin by serum and leucocytes occurs, especially in the corium and around the hair-follicles, sweat-glands and blood-vessels. As a result of this process there is a rapid destruction of the upper cells of the epidermis which results in desquamation.

The *mucous membrane* of the mouth and throat suffers an analogous inflammation with desquamation, especially well seen on the tongue and producing, in typical cases, the strawberry appearance. The inflammation, in average cases merely catarrhal, becomes in many instances pseudomembranous or even gangrenous in the fauces. The pseudomembranous inflammation of the throat exhibits various organisms,

¹ Discussion, Brit. Med. Journ., 1902, I, 777.

² Lancet, 1905, I, 273.

³ Lancet, 1864, II, 176.

⁴ Münch. med. Wochenschr., 1901, XLVIII, 1165.

⁵ Rilliet and Barthez, Mal. des enf., 1891, III, 164.

⁶ Boston City Hosp. Reports, 1899.

generally streptococci. It is probable that the presence of these germs is the evidence of a secondary infection. The faucial inflammation may spread to the nose and ears, and thence to the mastoid cells and even the meninges.

There is widespread involvement of the *lymphatic tissue* throughout the body. This is especially marked in the cervical lymphatic glands, which often suppurate, the pus containing streptococci. Swelling and edematous infiltration of the cellular tissue of the neck is common. The spleen is often enlarged and shows hyperplasia of the lymph follicles. The lymphatic tissue of the gastroenteric tract is hyperplastic, Peyer's patches being often much enlarged and prominent.

Endocarditis and pericarditis are not uncommon, and pleuritis and pneumonia may occur. Parenchymatous changes may be found in any of the organs of the body. These are not characteristic, but are due partly to the fever, as in all febrile diseases, partly to the septic involvement, and partly to the direct action of the scarlatinal poison. All these lesions, as well as the involvement of the kidneys so often present, are more conveniently considered under Complications.

Symptoms. TYPICAL SCARLET FEVER. Period of Incubation.—This period, between infection and the development of the first symptoms, is irregular within certain limits. In general it may be placed at from 1 day to 1 week, in by far the majority of cases from 2 to 4 days. Even periods of but a few hours have been recorded (Russeger).¹ On the other hand, periods of incubation lasting 12 days are not uncommon, and even as much as 20 days or more are on record (Hagenbach-Burckhardt).² As a rule the danger of developing the disease may be considered over after 7 days from exposure has elapsed. In cases of surgical scarlet fever, as for instance after tracheotomy, the period of incubation is usually very short. Severe cases also appear to have a shorter period of incubation than others.

Period of Invasion.—The onset is generally of great suddenness. Except in older children there is generally no distinct initial chill, although there may be chilliness and pallor. Convulsions are often among the first symptoms in quite young children. Vomiting, often repeated, occurs at the onset in the great majority of cases. Diarrhea occasionally accompanies it. The temperature rapidly rises to 103° or 104°F. (39.4° or 40°C.) or more, and the face becomes flushed. The pulse is rapid and there is headache and generally sore throat, of which, however, young children often make no complaint. The child looks and feels ill, sleeps badly, and is restless and decidedly prostrated. The lymphatic glands of the neck begin to enlarge. The eruption is now found in the throat (enanthem), giving a characteristic appearance; the mucous membrane, namely, of the hard and soft palate, the tonsils, and the anterior pillars of the fauces being unusually red, due to the presence here of closely packed minute dark-red macules. This condition rapidly spreads to the lining of the cheeks and gums. The macules may be distinctly visible or they may have fused to such an extent that only a uniform red flush is perceptible. The tongue is coated with the edges sometimes reddened. The tonsils are swollen and often exhibit whitish spots due to secretion retained in the follicles.

The duration of the invasion is short, from 12 hours or occasionally less up to 24 hours. It is of very common occurrence for a child to ex-

¹ Oester. med. Jahrb., 1848, LXIII, 277.

² Jahrb. f. Kinderh., 1886, XXIV, 105.

hibit the febrile symptoms and vomiting during the night and to be found with the eruption well developed by morning. A period of invasion lasting 36 or 48 hours and even longer is exceptionally observed (Fig. 51). In very mild cases there may be no prodromes whatever or none discovered.

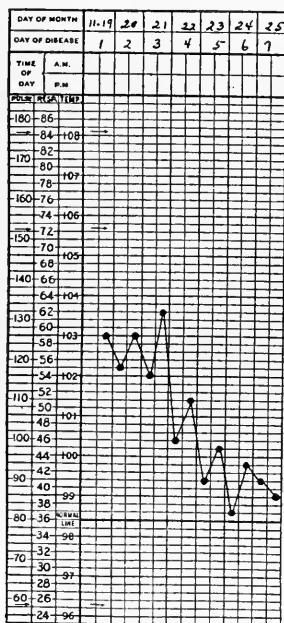


FIG. 51.

FIG. 51.—SCARLATINA. AVERAGE CASE EXCEPT FOR PROLONGED INVASION, WITH RASH ON THE 3D DAY.

Helen S., aged 2 years. Nov. 19, vomited, fever; Nov. 20, no complaint, throat very bright-red, suspiciously like scarlatina; Nov. 21, rash appearing, vomited; Nov. 22, general condition excellent, rash typical, abundant, throat less red; Nov. 23, rash fading, tongue red at tip, rash nearly gone.

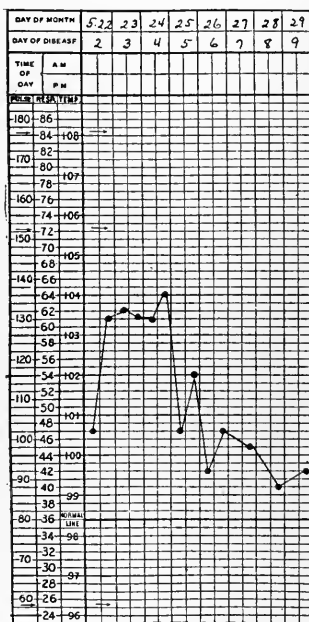


FIG. 52.

FIG. 52.—SCARLATINA. AVERAGE CASE.

Andreas L., aged 8½ years. May 21, nausea in evening, fever, sore throat; May 22, vomiting, diarrhea, secretion in tonsillar crypts, eruption on body; May 23, sore throat, restlessness; May 24, eruption very abundant; May 25, eruption dusker, tongue entirely denuded; May 27, eruption fading, throat and tongue sore; May 29, sore throat no longer complained of.

Period of Eruption.—The Rash.—The termination of the period of invasion is marked by the appearance of the rash on the skin. This develops first on the neck and upper portion of the chest; thence spreading rapidly to the rest of the trunk, the arms, and finally the lower extremities. Generally the face is little or not at all involved, the forehead and especially the circle about the mouth standing out prominently of a pale white color in contrast with the rest of the body; the cheeks being flushed, but not the seat of a truly punctate eruption. In some cases, however, there is an extensive development of eruption on the face. Viewed superficially the rash seems to be of a uniformly red color, but careful inspection shows it to consist of very minute, closely packed red points situated on a white base. Later the red hue of the punctæ grows dusker while the

white base takes on a shade of red as well. Though now more confluent the punctate character can still always be discovered. The eruption generally has spread to its full extent in 24 hours and often less. Sometimes, however, 2 or 3 days are required for this. Its greatest intensity of color may also be reached within 24 hours, but is not generally attained for 2 or 3 days. Where the integument is softest and finest the eruption is generally especially intense and most confluent. The skin is now hot, dry and somewhat swollen, the swelling being frequently particularly marked on the hands and feet. It often feels slightly rough to the hand rubbed over it. Decided itching is common. Drawing the fingernail over the red skin leaves for a moment a strikingly white line. This though very suggestive is not absolutely characteristic.

The rash maintains its intensity for from 1 to 3 days and then fades rapidly, following the order of its appearance. The total duration of the eruption is extremely variable. An average may be said to be from 3 to 7 days.



FIG. 53.—SCARLET FEVER.

Blotchy rash suggesting measles. (From a photograph.)

Normal Variations of the Eruption.—A number of variations occur in the eruption within the limits of the typical case. The color varies through many shades of red, from a pale rose to that of a boiled lobster, or to a deep red with a slightly brownish or bluish tint. When the eruption is not very extensive it may appear in smaller or larger blotches in some part of the body leaving other parts, especially areas on the extremities, entirely free (*Scarlatina variegata*). Sometimes it is macular in some portions and here strikingly suggests measles (Fig. 53). In many cases the skin is unusually rough, due to the presence of much infiltration in the minute red punctæ (*Scarlatina papulosa*), while in a large number there is a greater or less development of miliary vesicles (*Scarlatina miliaris*). These vesicles may occur in any part of the body. I have seen them so abundant that the scarlatinal color of the underlying skin was almost totally concealed¹ and the diagnosis made difficult. As a rule, however, they are few in number. Their occurrence does not appear to me to bear any relationship to the intensity of the ordinary scarlatinal rash, the amount of desquamation, or the severity of the case.

¹ Jacobi's Festschrift, 1900, 182.

In rare cases the vesicles may coalesce to a considerable extent, forming small blebs (*Scarlatina pemphigoides*).

In the so-called *Surgical Scarlet Fever* the rash develops sometimes first in the neighborhood of a wound, and thence spreads over the body, or appears in the ordinary sequence very soon after the receiving of a wound, it being supposed that the infection gained entrance through this. The symptoms do not differ materially from those of the disease as usually seen. A large number of cases are described in medical literature which would appear properly to belong in this category; yet very many others which have been called by this name are certainly rather instances of septic erythema. There is considerable doubt whether surgical scarlet fever exists as an entity, or is other than the mere coincidental occurrence of the disease in a surgical case.

Retraction of the Eruption.—It happens oftent hat the rash soon after its appearance suddenly fades, or, as it is commonly called, has “struck in,” the other symptoms persisting or growing worse. This disappearance is often the result of a feeble action of the heart. It is not the cause of unfavorable symptoms, but the accompaniment of them.

Other Symptoms of the Stage of Eruption.—The eruption of the mucous membrane of the mouth and throat (enanthem) is reaching its height when that of the skin begins to appear. The intense redness of the mucous membrane persists, and the tonsils, if not already affected, generally exhibit lacunæ filled with secretion, while swallowing is painful. The tongue begins to lose its white coating, and by the 3d to the 5th day of the disease has become entirely denuded, bright-red and with the fungiform papillæ swollen and prominent. The result is the “strawberry” or “raspberry” tongue. These titles have often been misapplied to the condition seen during invasion and in many other diseases, where the red papillæ, especially on the edges and tip, show prominently through the white coating. In many cases the tongue never becomes denuded throughout, yet the enlargement of the papillæ, at least of its tip and edges, is claimed by McCollum¹ to be invariably present and to constitute a valuable diagnostic sign in cases with doubtful or absent eruption. In typical cases the throat improves as the cutaneous rash and the other symptoms abate. By the 7th or 8th day of the disease the normal appearance of the tongue is nearly restored. Close examination, however, will show a persistence of some degree of redness and swelling of the papillæ at the edges and tip of the organ after other symptoms of the stage of eruption have entirely disappeared. The nose often exhibits a mucopurulent discharge. The lymphatic glands of the superficies of the body are found swollen, those of the neck, groins and axillæ and those below the body of the jaw, being most noticeably so. The spleen is often palpable. The *temperature* (Fig. 52) is subject to great variations, and no typical curve exists. As a rule it reaches its height from the 2d to the 3d day and continues at 102° to 104°F. (38.9° to 40°C.) with little variation between morning and evening, its elevation being generally in proportion to the severity of the attack. It begins to diminish as the eruption fades, falling by lysis, and reaching normal about the 9th or 10th day. Very often the elevation of temperature lasts a much shorter time than this. The pulse is rapid, often out of proportion to the elevation of temperature. This is generally considered one of the characteristics of the disease. The blood-pressure is sometimes reduced (Rolles-

¹ Boston City Hospital Reports, 1899.

ton)¹ but its condition possesses little practical value unless nephritis occurs. The bowels are usually not disturbed. There is thirst and loss of appetite. Vomiting is not common during the eruptive stage. Slight delirium may develop during the height of the fever. Either somnolence or restlessness may be present.

The *urine* is high-colored and diminished in amount and, if the temperature is high, may exhibit febrile albuminuria with cylindroids and possibly a few hyaline casts; but blood-cells and granular casts do not occur unless a complicating nephritis develops. Woody and Kolmer² found the diazo-reaction present in 8.53 per cent. of 375 cases examined. Urobilin is found in the majority of instances (Rach and Reuss;³ Gromski).⁴

The *blood* shows a moderate diminution in the number of red blood-cells and of hemoglobin. The original percentages are restored after several weeks. According to Tileston and Locke⁵ a hyperleucocytosis begins from the 2d to the 8th day reaching from 18,000 to 40,000 per c.mm. This falls gradually and reaches normal at the end of from 3 to 6 weeks. The polymorphonuclear cells are increased absolutely and relatively during the stages of invasion and eruption, reaching 85 to 90 per cent., and decrease to normal with the disappearance of the leucocytosis. The mononuclear cells are at first diminished, sometimes to even 4 or 5 per cent. The eosinophiles nearly or quite disappear, but rise above normal with defervescence and continue so into convalescence. These observations confirm for the most part the earlier studies of Kotschenkow,⁶ Saquepéc,⁷ Bowie,⁸ and others. Iodophilia is reported by Neutra⁹ and Magi.¹⁰ Great interest in recent years has attached to the discovery by Döhle¹¹ of certain "inclusion bodies" constantly found in the polymorphonuclear leucocytes of the blood in scarlet fever, as well as in the internal organs and lymphatic glands in fatal cases. Except in fatal toxic cases they are always present up to the 4th day. They were at first supposed to be microorganisms and believed to be the cause of the disease; but this view has been abandoned, and their nature is not known. Their absence is an indication that the condition is other than scarlet fever; but their presence is not specific, since they have been shown to occur, although in smaller numbers, in diphtheria, measles, lacunar tonsillitis and sepsis.

Period of Desquamation.—As other symptoms abate and the eruption fades the skin is left dry and rough and desquamation then begins, starting in the localities first invaded by the rash. Speaking very generally only, the process may be said to commence at the end of the 1st week of the disease. In many cases it is earlier, and in some much later. The scaling is of two forms. The first is a branny desquamation, somewhat similar to that seen in measles, and occurs on the head, neck and upper portion of the trunk. Upon the hands and feet there is a very

¹ Rolleston, Brit. Jour. Child. Dis., 1912, IX, 444.

² Arch. of Ped., 1912, XXIX, Jan.

³ Jahrb. f. Kinderh., 1910, LXXII, 422.

⁴ Przegl. pedyat. Ref. Monatsschr. f. Kinderh., Referat., 1914, XIII, 423.

⁵ Journ. of Infect. Dis., 1905, II, 375.

⁶ Wratch, 1891, No. 41. Ref. Jahrb. f. Kinderh., 1893, XXXVI, 109.

⁷ Arch. de méd. expériment., 1902, XIV, 101.

⁸ Journ. of Path. and Bact., 1902, VIII, 82.

⁹ Zeitschr. f. Heilk., 1906, XXVII, 433.

¹⁰ Gaz. degli Osped., 1908, XXIX, 433.

¹¹ Centralbl. f. Bakt. u. Parasit., 1892, XII, 909.



FIG. 54.—SCARLET FEVER.

Well-marked desquamation upon the dorsum of the hands and fingers, showing the lamellar peeling. (*Welch and Schamberg, Acute Contagious Diseases, 1905, 377.*)



FIG. 55.—SCARLET FEVER WITH UNUSUALLY SEVERE DESQUAMATION.

The enlarging scaling rings are well shown. (*Welch and Schamberg, Acute Contagious Diseases, 1905, 376.*)

characteristic lamellar peeling, the dead skin coming off in larger or smaller strips (Fig. 54), leaving a sharp contrast between the pink new skin exposed and the remaining old greyish-white skin. It begins here often at the tips of the fingers and toes and especially about the roots of the nails. Occasionally the skin is shed from the hands and feet in the form of true casts. The desquamation of the hands and feet is so characteristic that it is often possible to make the diagnosis on this appearance alone in cases not seen earlier in the attack. On the rest of the body and especially well marked on the trunk the branny desquamation begins as a small white scale which separates, leaving a pin-hole-like opening to the new skin beneath. The skin surrounding this gradually peels off, enlarging the pin-holes to wider circles which finally fuse (Fig. 55).

In some instances the two forms may be combined to a large extent throughout the body, except on the hands and feet where only the lamellar form occurs. Very frequently desquamation is absent or difficult to discover. This is especially true if the body has been bathed or oiled often. The scaling continues for from 10 days or less up to 2 or 3 weeks. It begins last and continues longest on the hands and feet, and here 4 or even 7 or 8 weeks may not see it completed. The average day of the disease on which the desquamation ceased in 91 cases in the Hospital for Scarlet Fever and Diphtheria of New York (1901) was the 47th.¹ Often it may not have commenced on the hands and feet by the time it is nearly or quite over on the rest of the body. Not infrequently after scaling has apparently ceased it recommences, and this process may be repeated several times. The duration and degree of desquamation usually bears some, but no necessary, relationship to the severity of the case and intensity of the eruption. There is reason to believe that it is even possible for desquamation to occur in regions where there has been no rash whatever; consequently other causes than the mere hyperemia of the skin would appear to be active agents in producing it. Nevertheless, as a rule, an intense eruption is attended by abundant desquamation, and vice versa.

Desquamation affects other epithelial structures as well. The nails exhibit it to a greater or less extent and the hair becomes brittle and falls. The teeth, too, are sometimes affected by the disease and exhibit erosions. In cases where miliaria has been extensive a fine branny scaling may take place early. This is not to be confounded with the true scarlatinial desquamation which occurs later. During the stage of desquamation all other symptoms are absent in normal cases and the patient feels well.

ATYPICAL TYPES OF SCARLET FEVER. (Mild; Severe; Anginose Malignant; Hemorrhagic).—Different classifications of the varieties of scarlet fever have been made. There are no strict boundaries separating one from another, but a convenient division is the following:

Mild Forms.—The attack may be unusually mild throughout, or some of the symptoms may be absent entirely. There may be a sudden onset with fever, which may disappear completely in the course of a very short time. The eruption may be so slightly developed that its nature is questionable, or it may be so evanescent or limited to so small an area that it is entirely overlooked. The throat may have exhibited only slight traces of redness, and the tongue may never have shown a decidedly characteristic appearance. The child may seem little, if at all, ill. In

¹ Report Hospital Scarlet Fever and Diphtheria Patients, 1901, 22.

many of these cases it is only on the occurrence later of desquamation, or perhaps of nephritis as a sequel, that a tardy diagnosis is made. Different varieties of the mild form are seen:

(a) In some cases the diagnosis from the beginning is unquestionable, but the disease runs a very short and mild course (Fig. 57), and the whole process is over in from 2 to 4 days, except perhaps for the desquamation later, although even this is often absent (*Abortive scarlet fever*).

(b) There seems to be no doubt that the eruption need not develop at all (*Scarlatina sine eruptione*). This is especially often seen in those persons associated with scarlet fever cases who have had the disease before. Such individuals exhibit only some fever and an angina with more or less involvement of the tongue. The condition has been denominated "*scarlatinal sore throat*." Such cases are as capable as others of spreading the disease.

(c) Another mild form shows an entire or nearly complete absence of fever (*Scarlatina afebrilis*). Such cases are generally accompanied by a very meagre development of the rash, and of other symptoms. Sometimes, however, the rash is very typical, yet with a temperature normal or rarely exceeding 100°F. (37.8°C.). I have seen this attended by an absolutely characteristic tongue and by subsequent nephritis, making the diagnosis certain (Figs. 58 and 59).

(d) In still another form inflammation of the pharynx is absent or represented only by a very slight redness of the pillars without swelling of the tonsils (*Scarlatina sine angina*).

The term "rudimentary form" is sometimes used to denote many of these mild cases with some symptoms undeveloped. It is however equally applicable to some of the severest atypical forms in which only certain of the symptoms of the disease are present although these are severe. The title is therefore confusing.

Severe Forms.—(a) In the ordinary severe form as most commonly seen all the symptoms of the disease may be present to an unusual degree.

The onset is rapid; there may be violent vomiting; the eruption is very intense and widespread and soon becomes dark red in tint; the temperature is persistently high; the glands of the neck are much swollen; the inflammation of the throat and mouth is severe; delirium is present; there is great restlessness; convulsions may occur and coma supervene; the pulse is rapid and weak; prostration is great and dyspnea may be marked. Death may take place in a few days, or the attack may last for weeks, the patient convalescing very slowly, if at all. The severity of the attack may appear to be the direct result of the scarlatinal poison, or the symptoms may be the varying ones of sepsis.

The severe forms of the disease show numerous variations from this type. These are not sharply differentiated from each other. Thus:

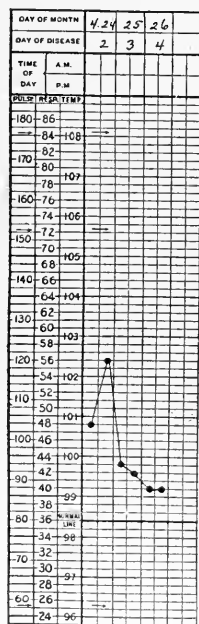


FIG. 57.—SCARLATINA; ABORTIVE FORM.

Weightman F., aged 9 years. Apr. 23, vomited; Apr. 24, rash appearing; Apr. 25, rash at height, well developed; Apr. 26, tongue beginning to peel; May 13, desquamation on hands and feet discovered; none found elsewhere.

(b) The severity may depend, perhaps, on the special prominence of one or a few symptoms or complications, the attack being "rudimentary" so far as the complete development of the disease is concerned.

(c) There occur differences in the development of the eruption. In many severe cases instead of being unusually well marked it is faint, localized, blotchy, and tends constantly to fade.

(d) In one variety the disease is characterized not so much by development of any localized symptoms as by long continuance of the fever without discoverable cause, there occurring such nervous symptoms

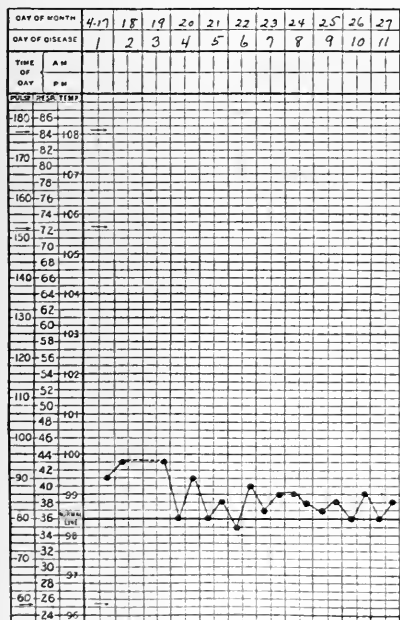


FIG. 58.

FIG. 58.—SCARLATINA AFEBRILIS.

Henry P., aged 9 years. Typical rash, slight redness of throat, moderate development of strawberry tongue. Later slight scarlatinal desquamation and albuminuria with casts.

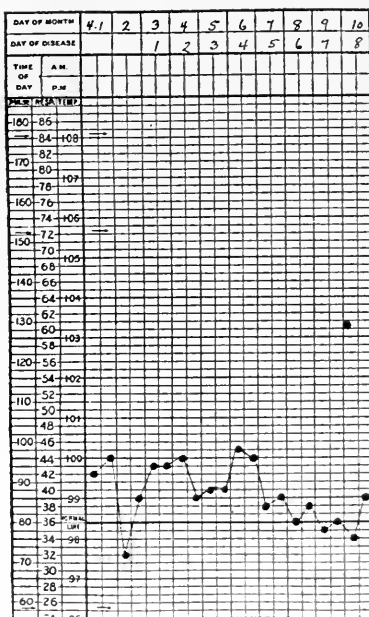


FIG. 59.

FIG. 59.—SCARLATINA AFEBRILIS.

Frank M., aged 3 years. Recovering from typhoid fever. Apr. 3, typical military scarlatinal eruption, tonsils red, secretion in follicles, tongue beginning to peel; Apr. 5, rash still very bright, miliaria drying; Apr. 8, profuse branny desquamation from the miliaria; Apr. 11, typical scarlatinal desquamation.

as headache, delirium, apathy, etc., or stupor and the symptoms in general of the typhoid state, on account of which the title *Scarlatina typhosa* has been applied to it.

(e) A fairly characteristic variety, dependent in reality on a complication, yet so common that it may well be described here, as is the anginose form (*Scarlatina anginosa*) (Fig. 60). In this the disease usually starts as an ordinary case and it is not until the 3d or 4th day or later that the characteristic symptoms appear. Sometimes the fever has commenced to fall, and the child seems about to convalesce, when, with an increase of temperature, the anginose symptoms begin. Pseudomembrane, the

result probably of invasion by streptococci, then develops on the tonsils and may spread to the uvula and pillars, the posterior wall of the pharynx, the nose, and occasionally the mouth. The submaxillary lymphatic glands become very much swollen. The appearance may be that typical of faucial diphtheria, and inasmuch as scarlet fever is so often complicated by a true diphtheritic invasion, the diagnosis can only be made by a bacteriological examination. In severe cases fever remains high, the pulse becomes weak, nervous symptoms are marked, and there develops a profound general septic state with great prostration. In other cases the lesions may go on to necrosis, and be attended by extensive sloughing with abscesses.

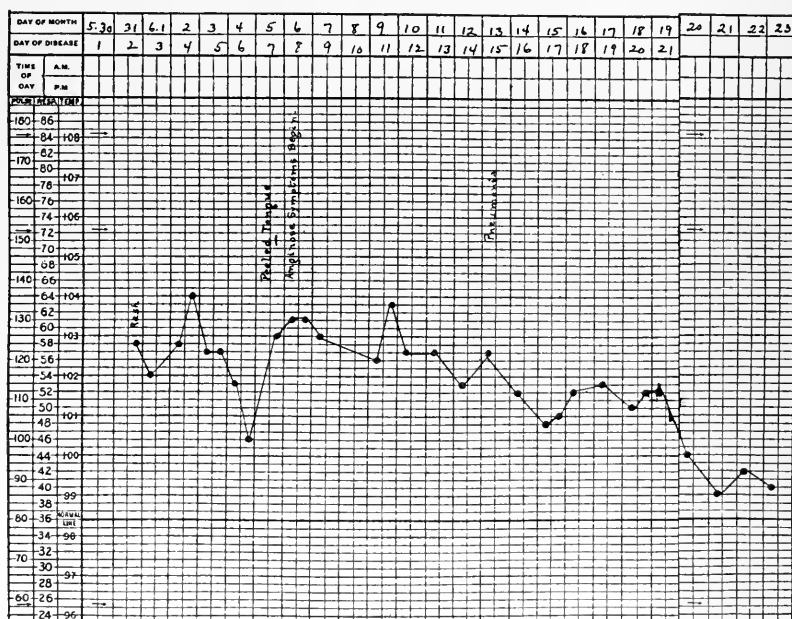


FIG. 60.—SCARLET FEVER FOLLOWED BY ANGINOSE SYMPTOMS AND LATER BY PNEUMONIA.

Minnie L., aged 7 years. May 30, vomiting in the evening and night; May 31, rash appeared, throat inflamed; June 2, throat more inflamed; June 5, tongue peeling; June 7, rash disappearing, inflammation of throat has increased, tonsils now thickly covered by white exudate; June 12, throat still inflamed, but better; pneumonia of low grade developed.

Even in the milder cases of anginose scarlet fever the local condition lasts for a week, more or less, and decidedly prolongs the attack while it adds greatly to the discomfort and debility of the patient and to the danger of involvement of the ears.

(f) One of the severe types of scarlet fever is the *malignant* or *cerebral variety* (*Scarlatina maligna*). The onset of this form is extremely sudden with a preponderance of nervous symptoms, the child appearing to suffer from an over-powering intoxication. There occur repeated vomiting, intense headache, very high fever, and restlessness and delirium soon passing into coma, perhaps with convulsions. The urine may be suppressed, the rash may be very intense, dark-red and often of a purplish cast from the presence of numerous petechiæ. Sometimes there are

hematuria, purpura, epistaxis, and hemorrhages from other parts of the body (Fig. 61) (*Scarlatina hemorrhagica*). In other cases the rash is but little developed, or even entirely absent. The inflammation of the throat likewise may not be present. In such cases there may be nothing on which to base a diagnosis, unless the case occur in connection with others. Death may take place in a few days, or even in a few hours. In still other instances the disease may begin in the ordinary manner and the evidence of malignancy does not appear until the 2d or 3d day.

Complications and Sequels.—Numerous disordered conditions may accompany or follow scarlet fever, but not many of these are of frequent occurrence. In 153,607 cases reported by the Metropolitan Asylums Board from 1900 to 1909 (Goodall)¹ the incidence of the more important complications equalled: otitis 13.1 per cent., adenitis 8.1 per cent., nephritis other than simple albuminuria 4.6 per cent., and arthritis 3.5 per cent.

Throat and Nose.—Different varieties of faucial involvement have been described, chief among them being the erythematous, pseudomembranous, and gangrenous forms. There exists, however, no sharp boundary line and they shade into each other. The erythematous throat, combined often with some engorgement of the tonsillar follicles with secretion, is always seen in typically normal cases. It is only when the process goes on to the development of a pseudomembrane, perhaps followed by extensive necrotic changes, that the condition can be considered as a complication. This occurs so often that it characterizes one of the variant forms of the disease. There may even be produced a condition which is clinically indistinguishable from that seen in diphtheria. My own experience has been that of others; that severe and even finally fatal cases of this condition may occur in which it is doubtful whether a scarlatinal rash has ever been present. The nose is often involved by the pseudomembrane, and an irritating thin discharge takes place from it which excoriates the upper lip. Nasal respiration may become impossible. Only occasionally is the larynx invaded. The symptoms attending this inflammation of the nose and throat have already been described (p. 321).

Ears.—Otitis media is one of the frequent complications. It is especially prone to develop when the inflammation of the throat has been severe, although by no means only then, the disease spreading along the Eustachian tube to the ear. Younger children are most disposed to it. The proportion of cases developing it varies greatly with the epidemic. Caiger² reports suppurating otitis media in 11.05 per cent. of 4015 cases;

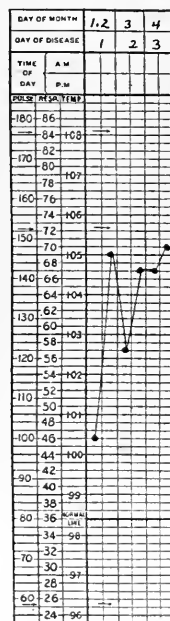


FIG. 61.—MALIGNANT SCARLET FEVER.

Domenick S., aged 2 years. Jan. 2, vomiting, dyspnea and irregular respiration; Jan. 3, red, uncharacteristic eruption, throat red, tonsils enlarged; Jan. 4, widespread, abundant petechial eruption combined with the scarlatinal rash, coma, respiration rapid and dyspneic, pulse rapid, tonsillar exudate; Jan. 5, death at 1 A.M.

¹ Garrod, Batten and Thursfield, *Diseases of Children*, 1913, 1009.

² Allbutt's *System of Med.*, 1897, III, 150.

Pugh¹ in 15 per cent. of 11,000 cases, and Borden² in 11 per cent. of 2232 cases and 454 autopsies. Baum³ found it in 1.07 per cent. of 628 cases although some pain in the ears was experienced in about 35 per cent. Both ears are generally affected. The greater frequency of perforative otitis in the severe cases of scarlet fever is shown by the analysis of Holmgren⁴ of 9590 subjects with this disease. In 511 fatal cases perforation had occurred in 36 per cent.

Otitis may terminate in complete recovery, generally after perforation, but many instances of some degree of permanent deafness owe their origin to a scarlatinal otitis. This occurred in 21.17 per cent. of Burkhardt-Meiringen's⁵ 85 cases of deafness. In older children with normal cerebral condition the otitis is generally accompanied by severe earache and deafness. In those, however, in which mental dullness exists, and in all younger patients, the symptoms are very obscure. Sometimes a return of fever with fretfulness is the only indication until spontaneous rupture of the drum-membrane occurs. The presence of tenderness over the region in front of the tragus or behind the ear is often the chief symptom unless the drum-membrane be frequently examined. The greatest watchfulness of these cases is therefore necessary. One of the chief dangers is the development of mastoid disease with subsequent purulent meningitis and septic symptoms. This may come on without the previous existence of otitis having been recognized at all. Even in cases where perforation of the drum-membrane has occurred the presence of a free flow of pus does not insure against the development of mastoiditis.

Otitis may occur either at the beginning of the disease or oftener as a sequel after desquamation has commenced or convalescence been entirely established.

Cervical Adenitis.—The lymphatic glands of the neck are always somewhat enlarged in all cases where sore throat is a prominent symptom. Sometimes they become greatly so, and may go in to the formation of abscess. This takes place oftenest as a sequel in the 2d week or later. It is attended by fever, but not by any marked septic symptoms, and is relieved by spontaneous opening or by incision. In bad cases of the anginose form, however, the engorgement and subsequent necrosis of the lymphatic glands is contemporaneous with the angina. The swelling may become extreme and a cellulitis of the tissues of the neck may develop, which can reach such an extent that the natural depression beneath the jaw is everywhere completely obliterated. The head is thrown back, and respiration may be much interfered with. This may go on to the formation of pus, which makes its way either toward the surface or along the sheaths of the muscles or the great vessels. Very extensive sloughing may take place, and blood vessels may be perforated. The attending symptoms are always of a gravely septic nature. Adenitis of a degree sufficient to be called a complication was found by Schick⁶ in 7.2 per cent. of 990 cases; and by Caiger and Dudgeon⁷ in 11.4 per cent. of 10,983 cases.

¹ Lancet, 1905, I, 273.

² Boston Med. and Surg. Journ., 1913, CLXVIII, 221.

³ Journ. Amer. Med. Assoc., 1903, Oct. 10, 906.

⁴ Otolaryngologiska Meddelanden, 1912, I, 1. Ref. Jahrb. f. Kinderh., 1913, LXVIII, 475.

⁵ Volkmann's Samml. klin. Vortr. Chir., No. 54, 1489.

⁶ Arch. f. Kinderh., 1905, XLIII, 459.

⁷ Loc. cit., 452.

Arthritis (Fig. 62).—This may often complicate or follow scarlet fever, generally in those past early childhood. Heubner¹ saw it in 8 per cent. of his 358 cases; Ashby² in 2.4 per cent. of 500 cases; Heiberg³ in 19.1 per cent. of 1000 cases; Baum⁴ in 3.7 per cent. of 628 cases, and Brade⁵ in 6.9 per cent. of 868 cases. In the majority of instances it is a synovitis (*scarlatinal rheumatism*) occurring about the end of the 1st week, sometimes earlier or later. It involves especially the hands, fingers and elbows, although any of the joints may be affected. The attack lasts generally

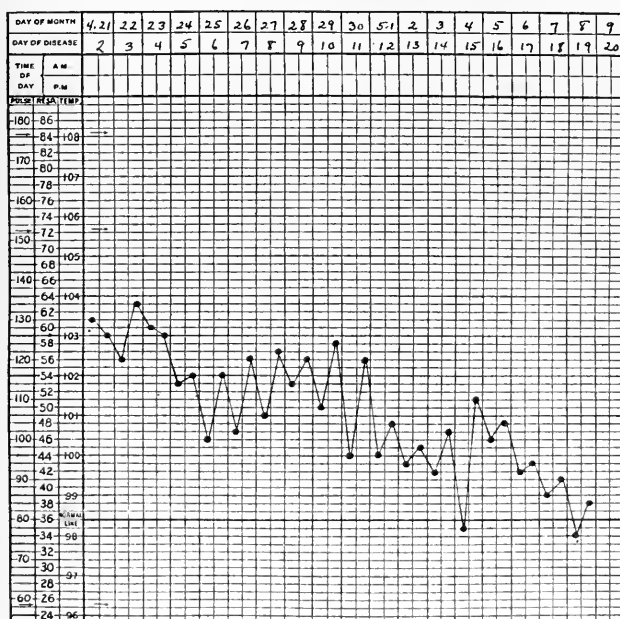


FIG. 62.—SCARLET FEVER WITH TEMPERATURE PROLONGED BY ARTHRITIS.

Genette F., aged 5 years. Apr. 20, fever, vomiting; Apr. 21, well-developed eruption; Apr. 22, a few white spots on tonsils; Apr. 23, rash at maximum, tongue peeling; Apr. 24, rash fading; Apr. 26, multiple arthritis, involving fingers, elbows, knees and ankles; May 5, joints improving.

not more than 3 or 4 days. It is a mild affection characterized by pain and with generally moderate swelling and redness. There may, however, be nothing visible on inspection of the joints. The likelihood of its developing bears no relationship to the severity of the scarlatinal attack. It is probably not truly rheumatic in nature, although cases are occasionally seen in which true rheumatism and scarlet fever appear to be combined.

Another less frequent form of arthritis is apparently septic in nature and goes on to suppuration. It may involve one or more of the larger joints, and possesses a graver prognosis.

Nephritis.—This is one of the most common and often most serious of the complications or sequels of scarlet fever. Some writers regard

¹ Deutsche Klinik, 1902, VII, 286.

² Brit. Med. Journ., 1883, II, 514.

³ Zeitschr. f. Hyg. u. Infektionskrankh., 1910, LXV, 237.

⁴ Journ. Amer. Med. Assoc., 1903, Oct. 10, 906.

⁵ Inaug. Dissert. Leipzig, 1904. Ref. Monatsschr. f. Kinderh., 1904, III, 348.

a degree of involvement of the kidneys as one of the constant symptoms of the disease. The presence, however, of slight albuminuria, perhaps with cylindroids and even hyaline casts, as often seen in the first days of an attack of scarlet fever when the temperature is high, is what is liable to occur in any of the febrile infectious diseases, and cannot properly be looked upon as evidence of nephritis. In the severer cases of scarlet fever, however, particularly when any septic process is present, as for instance in the throat, an acute diffuse nephritis frequently develops during the height of the disease. It is seen with great frequency in the malignant cases. The nephritis may come on with suddenness and severity, with great diminution of the urinary secretion and the presence in it of much albumin and with casts and blood. The symptoms are those of uremia rather than of dropsy. Convulsions often occur. In cases not so severe there may be no special symptoms, or those present may be masked by the graver ones of the scarlet fever itself.

The most frequent form of nephritis, however, is that denominated *post-scarlatinal*, occurring as a sequel. This develops in the 3d or 4th week after the onset of the scarlet fever, and is just as liable to follow the mildest cases as any others. The lesions are oftenest those of a glomerular nephritis. The onset may be sudden with widespread dropsy and with the very scanty secretion of a smoky or coffee-colored urine containing much albumin, blood cells, granular epithelium and very numerous casts of different forms. Convulsions, vomiting and other uremic symptoms may develop. Complete anuria may last for a time (Northrup¹ 5 days; recovery). Death may follow promptly, or there may be a gradual amelioration of the symptoms, the urine becoming more abundant and of lower specific gravity, the fever lessening, and the child showing evidences of anemia. In the majority of cases, however, the symptoms are much less severe and their onset often insidious. There may be only slight edema, especially of the hands, feet and eyelids; a moderate degree of fever, irritability and restlessness; and but a small amount of albumin in the urine with a few casts of different sorts. As in the severer but not fatal cases, convalescence is slow, the albumin and casts continuing perhaps for months and finally disappearing, and recovery being complete in the majority of instances. There is noted a decided tendency for the nephritis to relapse, and this may persist for many months. There have been instances reported, and even some epidemics, in which edema has developed subsequent to scarlet fever yet without any evidence in the urine of an inflammation of the kidneys (Quinke).² In some such cases, however, the lesions of nephritis have been found *post-mortem*. In only a few instances does the nephritis become chronic. A study by Rosenfeld and Schwitka v. Rechtenstamm³ of 94 individuals who had had acute scarlatinal nephritis several years previously failed to reveal a single instance of severe chronic nephritis remaining.

Nephritis may, of course, lead to other complications, such as cardiac hypertrophy and dilatation, effusion into the pleural and pericardial sacs, and the like. The relative frequency of the occurrence of nephritis in connection with scarlet fever varies greatly with the epidemic. The views of writers as to what constitutes the evidence of nephritis also influence the statistics. Ashby and Wright⁴ estimate the average propor-

¹ Med. Rec., 1910, LXXVIII, 706.

² Berliner klinische Wochenschrift, 1882, XIX, 57.

³ Zeitschr. f. Kinderh., Orig., 1912, IV, 265.

⁴ Diseases of Children, 1893, 247.

tion of patients attacked as 6 per cent. Caiger and Dudgeon¹ give 4 per cent. in 10,983 cases; Royer² 7.76 per cent. of 756 cases. Others have given higher figures.

The causes of nephritis in scarlet fever are not certainly known. In the cases associated with septic processes some pus-germ is probably the active agent. In those developing during malignant scarlet fever and in the post-scarlatinal cases it is probably the toxic products of the scarlet fever germ, whatever this may be. The predisposing influence of external conditions is uncertain. (Fuller reference to the pathology and treatment of scarlatinal nephritis will be found under the heading of Nephritis).

Circulatory System.—Functional transitory disturbances of the heart are not uncommon during scarlet fever, but serious lesions directly produced by the disease are less frequent. Endocarditis and pericarditis occur occasionally as the result of a complicating nephritis or a rheumatic synovitis, or as one of other septic manifestations. I have observed very few cases in which chronic valvular disease could be without question attributed to an antecedent scarlatina. Embolism of the pulmonary or other arteries has been observed. Myocardial degeneration is present to some extent in many cases, especially if these are severe and accompanied by high fever. If considerable, it may produce acute dilatation. Dilatation, however, and especially hypertrophy, generally depend upon a nephritis and are sequels rather than complications. Hypertrophy the result of this cause comes on rapidly and may present demonstrable physical signs even within a week from the onset of the renal inflammation. Popischill³ believed that the heart is often characteristically affected in scarlet fever, there being a splitting of the first sound and a murmur suggesting pericarditis; these depending upon myocardial changes. The symptom is observed especially at the beginning of the attack. Lederer and Stolte⁴ confirm this observation.

Respiratory System.—The general opinion is that pulmonary complications and sequels are not nearly so frequent in scarlet fever as in some other diseases. Hutinel,⁵ however, found that about $\frac{1}{3}$ of the deaths in 2500 cases were from bronchopneumonia, and that croupous pneumonia and pleurisy also were frequent complications or sequels. The involvement of the nasal mucous membrane has been spoken of in connection with the throat. Pseudo-membranous laryngitis not associated with the germs of diphtheria is of rare occurrence. Pleurisy with effusion, often purulent, is an occasional sequel. Its onset is frequently very insidious and entirely unsuspected. Bronchitis occasionally develops. Bronchopneumonia is a dangerous complication, usually occurring only in association with nephritis. Severe septic cases of scarlet fever quite commonly exhibit bronchopneumonia at autopsy. Croupous pneumonia is generally believed to be uncommon except in combination with nephritis. The opinion of Hutinel opposes this. Edema of the lungs and edema of the glottis occasionally occur as a result of disease of the kidneys.

Gastrointestinal Complications.—These are not, as a rule, troublesome. Vomiting is very common at the beginning of the attack, and

¹ *Loc. cit.*, 2, 45

² *Penna. Med. Journ.*, 1906-7, X, 286.

³ *Wien. klin. Wochenschr.*, 1907, XX, 1089.

⁴ *Jahrb. f. Kinderh.*, 1911, LXXIV, 395.

⁵ *Arch. de méd. des enf.*, 1916, XIX, 57.

ulcerative stomatitis is a complication occasionally seen in young children. It is usually mild, but sometimes as a sequel is severe. Diarrhea is a frequent complication. It is generally of a catarrhal nature, but may be inflammatory. In some cases it is very obstinate and debilitating. It may develop during the height of the disease, or be one of the earliest symptoms of the eruptive stage. Slight icterus is very common.

Nervous System.—Among nervous complications and sequels are especially to be mentioned repeated convulsions. At the onset of the attack these generally have little significance, but later they are suggestive of uremia. Meningitis and thrombosis of the cerebral veins or sinuses may occur as sequels depending upon an otitis. Occasionally a meningitis occurs earlier in the disease and independently of any affection of the ear. Hemiplegia has been known to follow meningeal hemorrhage or be caused by embolism dependent upon an endocarditis. Paralysis has also been seen as a result of neuritis or of disease of the spinal cord. Chorea and epilepsy are sequelæ occasionally reported, and various psychoses are on record. Amaurosis may develop as a result of nephritis.

Cutaneous Complications.—Urticaria and erythema are sometimes observed after the disappearance of the scarlatinal eruption, and eczema is not an uncommon sequel. Rolleston¹ found herpes facialis 27 times in 413 cases. Symmetrical gangrene is an occasional complication. Silberstein² added 1 case to 13 others collected from medical literature.

Other infectious diseases may occur in combination with scarlet fever or as sequels to it. One of the most frequent and serious combinations is that of scarlet fever and diphtheria, the latter developing generally in a patient already suffering from the former. It is more prone to appear from the 3d to the 6th week after the attack of scarlet fever, sometimes earlier or later. Measles and scarlet fever have often occurred together. Among other combinations are those with varicella, variola, pertussis, erysipelas, typhoid fever, etc. Either disease may develop first. Tuberculosis already in the system may be brought into activity by the occurrence of scarlet fever.

Among other rarer complications or sequels which have been reported may be mentioned perichondritis of the larynx; esophagitis; necrotic inflammation of the stomach; gastric hemorrhage; optic neuritis; icterus; encephalitis; periostitis; peritonitis; noma; furunculosis; vulvo-vaginitis; purpura; myositis; pemphigus; and glycosuria.

Relapse.—Relapse is occasionally seen, but is rare. McCollum³ found it 4 times in 1000 cases (0.4 per cent.) and Sloan⁴ 154 times in 14,143 cases (1.8 per cent.) of the Hospitals of the Metropolitan Asylums Board. It consists in the return of some or all of the symptoms after the disease is apparently over, but while the original infection is doubtless still in the system. The mere reappearance of the eruption after a brief fading is not to be classified here; nor is the simple return of temperature an indication of relapse, since this often occurs, sometimes without the discovery of the cause being possible. There must be a combination of symptoms sufficient to justify the diagnosis of a true relapse. Relapse is seen oftenest in the middle or at the end of the 4th week and may be either milder or more severe than the primary attack. Lettry⁵ in analyz-

¹ Brit. Journ. of Dermatol., 1910, XXII, 309.

² Jahrb. f. Kinderh., 1912, LXXV, 350.

³ Boston City Hosp. Reports, 1889.

⁴ Lancet, 1903, I, 436.

⁵ Thèse de Paris, 1906-7.

ing 46 collected instances found relapse oftenest between the 15th and 35th days.

Recurrence.—Recurrence of the disease in the form of a distinct second attack after a considerable interval is of great rarity. One attack usually protects from subsequent ones. One hears of instances frequently, especially from the laity, but most of these are certainly mistakes in diagnosis. Thomas¹ reports a number of undoubted cases collected from medical literature, but has himself observed it but once in hundreds of cases, and Hensch² saw it but once. J. McCrae³ reported 9 instances said to be second attacks in 850 cases (1 per cent.), and Weissenberg⁴ 7 cases. I have treated one child with 2 distinct and undoubted attacks separated by an interval of a year.

In speaking of recurrence no reference is intended here to that which might with some propriety be called this; viz. the development of a scarlatinal angina in exposed adults who had previously had the disease.

Prognosis.—The continuously mild cases nearly invariably recover; yet cases beginning in this way may later become severe, or a dangerous complication or sequel may arise. The prognosis in every instance must, therefore, be always most guarded throughout. Only after all danger of sequels is over can one feel at ease. The mortality appears to vary greatly with the epidemic and is different in different countries. Thus in 11,216 cases reported in Philadelphia during 5 years the mortality was 5.4 per cent. (Graham);⁵ and in 1072 cases reported in 1915, 2.4 per cent. (Ostheimer).⁶ Of 21,834 cases occurring in Hamburg during 10 years 6.8 per cent. died (Reinke).⁷ Of 15,137 cases occurring in Stockholm during 21 years 16.3 per cent. died (Carlson)⁸ the lowest yearly mortality being 2.8 per cent. and the highest 28.8 per cent. Of 1598 cases in the Manchester Children's Hospital during 10 years 11.8 per cent. died (Ashby and Wright).⁹ In 1000 cases in the Boston City Hospital the mortality was 9.8 per cent. (McCollum).¹⁰ Of 84,380 cases in Norway collected by Johannessen¹¹ 14.17 per cent. died, while of 167,840 cases in the Metropolitan Asylums Board's Hospitals during 13 years but 4.3 per cent. died (Caiger).¹²

These figures show the great variation in statistics, but on the whole there appears to have been a decidedly decreasing mortality, scarlet fever being by no means as serious an affection as it was fifty or more years ago. Roughly speaking the general mortality may be said to vary from 10 per cent. to 15 per cent., although it is much less in uncomplicated cases. It is the general experience that in private practice among the better classes the death-rate is decidedly less than this. As compared with the mortality from other causes that from scarlatina varies from about 2 per cent. to 6 per cent. or more of the total deaths.

Certain conditions, however, influence the mortality very unfavor-

¹ Ziemssen's Handb., 1874, B. II Th II, 176.

² Kinderkrankheiten, 1895, 675.

³ Canadian Med. Assoc. Journ., 1911, 1, 293.

⁴ Arch. f. Kinderh., 1909, LII, 17.

⁵ Jour. Amer. Med. Assoc., 1917, LXVII, 1272.

⁶ Amer. Journ. Pub. Health, 1916, VI, 1104.

⁷ Bericht d. Medicinalraths, etc., 1894, 58. Ref. v. Jürgensen, Nothnagel's Encyclopedie of Pract. Med., Scarlatina, 603.

⁸ Ref. v. Jürgensen, *loc. cit.*, 603.

⁹ Dis. of Child., 1893, 240.

¹⁰ Report Boston City Hospital, 1899.

¹¹ Die epidem. Verbreitung d. Scharlachfieber in Norwegen, 1884.

¹² Allbutt and Rolleston, Syst. of Med., 1906, II, 1, 429.

ably. Age is a prominent factor here, the disease being much more fatal in children under 5 years. Seitz¹ found the mortality 40.6 per cent. in the 1st year of life. In general the younger the patient the greater is the danger of death. The development of severe nephritis during the attack greatly increases the death-rate. Post-scarlatinal nephritis does so to some extent, although to a much less degree. Symptoms of sepsis, especially those dependent on severe involvement of the throat, are unfavorable. In proportion also to the intensity of the action of the scarlatina toxin the danger grows. Hence malignant cases are nearly invariably fatal. A very rapid pulse, an unusually high temperature, evidence of decided involvement of the nervous system or of the throat, and an irregularly or poorly developed eruption if accompanied by severe symptoms all point to the existence of a grave case.

Diagnosis.—This is not difficult in typical cases, and rests chiefly on the sudden onset with vomiting, high fever and sore throat, followed by the rapid development and characteristic spread of the punctate eruption, and later the peculiar desquamation. In very mild cases, however, as well as in the rapidly malignant ones, the diagnosis is often extremely difficult or even impossible. In the latter the rash may be entirely uncharacteristic or even absent. In the former it may be limited to a small area, poorly developed or transitory and entirely overlooked, the tongue and throat may not present typical changes, and desquamation may not be discovered. Other cases may present only the affection of the throat without the cutaneous eruption. In some instances no desquamation can be found at any time, especially if inunction has been employed. In Negroes the diagnosis is often very difficult, owing to the concealment of the eruption by the dark hue of the skin. In some such instances only the occurrence of nephritis, otitis, or other sequel, or of the characteristic desquamation, makes the diagnosis clear. It is the clinical picture as a whole rather than any one symptom upon which the diagnosis must be based.

As an aid to the recognition of the scarlatinal eruption Pastia² described a uniform linear redness in the transverse fold of the skin at the elbow, more intense than the scarlatinal eruption. It is present at the very beginning of the eruptive stage and lasts after the rash has faded. It appears to be produced by capillary hemorrhages. Its presence in scarlet fever and its absence in measles is confirmed by Bizzarri³ and by Lippmann.⁴ The Rumpel-Leede sign⁵ is another supposed evidence of the existence of scarlet fever. It consists in the development of punctate hemorrhages into the skin of the elbow-fold after compression of the upper arm has continued from 5 to 20 minutes. It is claimed to be especially applicable early in the disease. Experiments by Michael⁶ showed that a positive sign could be elicited in normal children and was, therefore, no proof of the existence of scarlet fever. Leede believed that a negative result excludes scarlet fever.

Certain diseases are especially to be distinguished from scarlet fever. *Rubella* presents at times the closest possible resemblance, and even careful and repeated observation may not solve the question in isolated cases,

¹ Münch. med. Wochenschr., 1898, XLV, 76.

² Arch. de méd. des enf., 1911, XIV, 130.

³ La Pediatria, 1912, XX, 898.

⁴ Pediatrics, 1912, XXIV, 358.

⁵ Rumpel, Münch. med. Wochenschr., 1909, LVI, 1404; Leede, Münch. med. Wochenschr., 1911, LVIII, 293; 1673.

⁶ Arch. of Ped., 1912, XXIX, 298.

or even in localized epidemics. The absence of general symptoms and of scarlatinal changes in the mouth and throat aid in its recognition. So, too, there may nearly always be found somewhere on the body the characteristic macular eruption of rubella, even when the greater part of the surface is covered by a scarlatiniform rash. (See Rubella.) Yet I do not know of other two affections where differential diagnosis can be at times the source of more perplexity. *Measles* seldom causes any confusion. When it presents a poorly developed eruption it may, however, be distinguished from some cases of scarlet fever only with the greatest difficulty. The longer incubation and invasion, and the catarrhal symptoms of the latter; the buccal eruption and the slower development of the rash are suggestive.

Diphtheria, if attended by an erythema in the early stages, may not be at first distinguishable from scarlet fever with severe anginous symptoms. The difficulty arises, too, in the cases of scarlet fever in which no rash was discoverable. The close study of the sequence of symptoms will often be of service. The rash in diphtheria is not very frequent, and is generally confined to the trunk. The discovery of the Klebs-Loeffler bacilli would settle the question, were it not that the two diseases may be combined. Consequently a positive diagnosis is sometimes impossible.

Scarlatiniform Erythema is one of the most puzzling conditions so far as diagnosis is concerned. This erythema may be due to sepsis, other infectious diseases, various medicaments, or to acute desquamative dermatitis. It is not infrequent in infants with slight disturbance of digestion. Sepsis is the cause in diphtheria, and also probably in the majority of the cases called "surgical scarlet fever." The rash resembles that of scarlet fever in appearance, and may be followed by desquamation. The history of the case, the failure of development of the eruption in the usual sequence and extent, and the absence of the other symptoms of scarlatina aid in the diagnosis. Typhoid fever, grippe and varicella occasionally exhibit an erythema early in the attack and this may make the diagnosis at first obscure. The absence of the scarlatinal symptoms, the transitory character of the rash, and the appearance later of the other manifestations characteristic of these different diseases remove the difficulty in diagnosis. The prodromal erythema of variola is distinguished by its peculiar localization. Various erythemata caused by drugs are at times perplexing. Among these are especially those due to quinine, chloral, salicylic acid compounds, belladonna, antipyrine, and animal sera, especially the diphtheria antitoxin. Local irritants such as mustard-plasters have often been the source of confusion. The occurrence of the atropine eruption has especially been a cause of alarm to parents. In none of these are the other symptoms of scarlet fever present, except that occasionally the eruption may be accompanied by fever and vomiting. Quinine will sometimes produce a scarlatiniform eruption followed by very characteristic scarlatiniform desquamation. Acute desquamative dermatitis (recurrent exfoliating dermatitis) is a peculiar affection which may resemble scarlet fever very closely. Probably cases supposed to be second and third attacks of scarlet fever are really instances of the disease. It is attended by fever, a widespread erythematous eruption, and often by desquamation which may even occur in the form of casts of the skin of the hands and feet, as in some cases of scarlet fever. In these respects it cannot be distinguished from scarlet fever. Pharyngeal symptoms are, however, absent.

Treatment. (A) **Prophylaxis. Quarantine.**—Every case of suspected scarlet fever should be isolated at once, and kept so until all danger of the infection of others is over. The attempt to limit the spread of the disease offers much greater chance of success than in the case of some of the other infectious fevers. How long the quarantine should last is uncertain. At least 6 weeks should be the rule, and longer than this if desquamation or purulent discharge from the ears or nose is present. This is in view of the infectiousness which possibly inheres in the contaminated scales and certainly in the discharges. The studies of a number of observers, however (Millard;¹ Lauder),² indicate that this time is unnecessarily long; and that 4 or 5 weeks insures as great safety as 6, provided there is no abnormal discharge from the nose or ears. Priestly³ sent home from the hospitals 120 individuals still desquamating; with no development of the disease following in other members of the families. These cases, however, had been anointed systematically with a disinfectant. The general methods for preventing the spread of the disease, the selection of the room and care of the patient, and the management of other children in the family, are described under General Management of Infectious Diseases (p. 306), and must be strictly followed. Preventive inoculation was attempted years ago by various physicians without satisfactory results. In recent years similar efforts have been made and good results reported. Gabritschewsky⁴ employed a form of preventive vaccination in about 50,000 children and claimed success. This has been confirmed by some observers, but found without value by others (Kolmer).⁵

(B) **Treatment of the Attack.**—There is no method known which curtails the attack in any way. The treatment is purely symptomatic. The patient should be kept absolutely in bed until desquamation is over. The confinement to bed should continue for at least 3 weeks even in the mild cases. The diet should if possible be liquid until the temperature is normal, milk being by far the best food. Where, as is often the case with small children, the appetite is lost and milk is refused, some of the cereal foods may be tried early, and the diet varied considerably. After fever has ceased a diet of cereals and milk is always in order, reserving meat and eggs until the end of at least the 3d week of the disease. This plan of treatment throws less work upon the kidneys. The injuriousness of meat in this disease has been, however, disputed. Gerstley⁶ in a trial upon 306 cases, half of them given the ordinary full diet, could see no difference in the incidence of nephritis. These results confirm the observations of Popischill and Weiss,⁷ but are opposed to the opinion and practice of the majority of pediatricists. Water to drink should be administered freely. Daily warm ablution should be given to every child for the sake of cleanliness and comfort, with proper precautions against exposure, washing being done under the bed-clothes, or with the uncovering of but one portion of the body at a time.

The room should not be hot, and fresh air is an essential, but draughts must be carefully avoided during the apyretic period of convalescence. Much has been written of the innocuousness of exposure to draughts of air in this disease. While it is undoubtedly true that infection is the

¹ *Lancet*, 1902, Apr. 5

² *Lancet*, 1904, I, 712.

³ *Transac. Epid. Soc.*, 1894-5, XIV, 71.

⁴ *Ref. Wladimiroff, Arch. f. Kinderh.*, 1909, LII, 28.

⁵ *Arch. Int. Med.* 1912, IX, 220.

⁶ *Monatsschr. f. Kinderh., Orig.*, 1913, XII, 121.

⁷ *Ueber Scharlach*, 1911. *Ref. Gerstley, loc. cit.*

active cause in the production of nephritis and other complications, there seems no good reason why surface-chilling may not act as a predisposing cause, and why no precaution should be taken against it. The bed-covering should be light, the sensations of the patient, when these can be ascertained, being the best guide. The skin should be kept oiled with petrolatum or a weak carbolized oil (2 per cent.) or, in the case of young children, with a 1 per cent. thymol ointment. This allays the itching and prevents also the dissemination of the scales, at the same time disinfecting them, if they have become soiled by mucus or purulent secretion, and favors a more rapid desquamation. The mouth, throat, and nose should be examined daily in order that appropriate treatment may be commenced at once if indicated. (See p. 335.) The employment of mild antiseptic gargles and sprays aids in preventing the development of any serious trouble here. The urine also should be examined frequently.

Little internal medication is needed. Stimulation is not required in ordinary cases. A mild diuretic, such as citrate of potash, is of advantage in maintaining diuresis without irritation of the kidneys. Various drugs have at different times been recommended as specifics. None of these have proven of any certain value.

Much has been written regarding the *serum-treatment* of scarlet fever, but without any generally accepted conclusions. Marmorek's¹ anti-streptococcic serum has been tried in scarlet fever on the ground that a streptococcus was the cause of the disease. In Baginski's² experience this proved of doubtful service. Later a serum was advocated by Aronson,³ obtained by inoculation of horses with streptococci from scarlet fever patients after repeated passage of the cultures through animals by inoculation. The value of this serum has not yet been satisfactorily demonstrated. Moser⁴ produced a polyvalent serum in a somewhat similar manner using, however, cultures from the blood of scarlatinal patients, and inoculating horses without previous strengthening of the toxins by animal transmission. He claimed excellent results with this, and these have been supported by many investigators, as Escherich⁵ and many others, while Heubner,⁶ Ganghofner⁷ and others obtained no favorable results. (For a careful review see article by Fedinski.)⁸ If scarlet fever is dependent upon a streptococcus, which has not as yet been proven, it is evident that good results from any anti-streptococcic serum can be hoped for only in cases where the danger is from the septic manifestations. Under such circumstances the treatment may well be tried. The serum, if given in large doses, 100 to 200 c.c. (3.4 to 6.8 fl.oz.) at a time, and in the first 3 or 4 days, offers the greatest likelihood of doing good. Leyden⁹ viewed favorably the employment of blood-serum of patients convalescing from the disease, and Landsteiner and Levaditi,¹⁰ and Zingher¹¹ report surprising results some-

¹ Ann. de l'inst. Pasteur, 1896, X, 47.

² Berl. klin. Wochenschr., 1896, 340.

³ Verhandl. Berl. med. Ges., 1902, XXXIII, 253.

⁴ Jahrb. f. Kinderh., 1903, LVII, 1.

⁵ Wien. klin. Wochenschr., 1903, XVI, 663.

⁶ Berl. klin. Wochenschr., 1904, XLI, 373.

⁷ Deut. med. Wochenschr., 1905, Apr. 5, 529.

⁸ Jahrbuch f. Kinderh., 1910, LXXI, 89.

⁹ Münch. med. Wochenschr., 1902, 159.

¹⁰ Ann. de l'inst. Pasteur, 1911, XXV, 754.

¹¹ Journ. Amer. Med. Assoc., 1915, LXV, 875.

times seen in severely ill patients. Not less than 50 c.c. (1.7 fl.oz.) should be given intravenously or into the muscles.

The treatment of some of the more important symptoms and complications may be considered separately:

Fever.—A temperature of 104°F. (40°C.) lasting for a short time is generally a matter of no special consequence. When, however, high fever is prolonged and is accompanied by nervous symptoms, treatment is required. Hydrotherapeutic measures are generally much to be preferred to internal medication. Sponging with luke-warm water or with alcohol and water may be employed. If this is not sufficient, sponging with water at a temperature of 70° to 80°F. (21.1° to 26.7°C.) may be tried, or in urgent cases cooler than this. Great discretion must be exercised, however, in young children and especially in infants, since these often do not bear cold water well in any febrile disorder. Every case must be treated as an individual and all depression of pulse-strength assiduously avoided. Very often submersion in a tepid bath of 85° to 90°F. (29.4° to 32.2°C.) or even in a warm bath of 90° to 100°F. (32.2° to 37.8°C.) will effect decided reduction of temperature. In very urgent cases the graduated bath may be employed, the temperature of the water after the child is in the tub being gradually reduced to even 70°F. (21.1°C.); rarely lower than this. The warm or cold pack frequently repeated is often very useful and better tolerated than the bath. Hydrotherapy must always be used with especial caution, if at all, where decided cardiac weakness exists. When, too, good reaction does not take place, or when a young child fights violently against the treatment, it may sometimes do more harm than good.

The employment of antipyretic drugs of the coal-tar series for the reduction of temperature is only exceptionally indicated. There are times, however, when they are very serviceable, when for any reason hydrotherapy cannot be employed. They should be given in small doses frequently repeated.

Nervous symptoms attending high temperature are often much benefited by hydrotherapy. Indeed, it is the combating of these, rather than the simple reduction of temperature, which is the chief aim of hydrotherapeutic measures. An ice cap to the head is frequently of advantage under these circumstances. The coal-tar derivatives employed to combat nervous symptoms have a value much greater than that of the mere reduction of fever which they effect. Their administration is often followed by the relief of delirium, stupor, jactitation or a convulsive condition, which is greater than can be attributed to the fall of temperature. My own preference is for phenacetin or antipyrine. It is important to give repeated small doses rather than a single larger one, since a decided fall of temperature may be attended by prostration. In the majority of instances hydrotherapy is to be preferred. For the convulsions developing during scarlatinal nephritis both Allaria¹ and Sheffield² have seen benefit from the employment of lumbar puncture.

Cardiac weakness, as shown by the rapid and feeble or irregular pulse, weak heart-sounds, or persistent coldness and cyanosis of the extremities, demands the prompt and energetic use of stimulants. Even before these symptoms have actually made their appearance, but where, as in septic cases or those with prolonged high temperature, it is feared that weakness of the heart may develop, stimulants may well be employed.

¹ *Gaz. degli. osp. ed del. clin.*, 1911, XXXII, 1291.

² *Pediatrics*, 1912, XXIV, 99.

Digitalis, strychnine and alcohol by the mouth, or camphor and caffeine given hypodermically, may be selected according to the demands of the case.

Inflammation of the throat and nose when mild requires no special medication. When more severe, as in anginose cases, it often demands active treatment, the nature and frequency of which varies with the case. The nose may be sprayed or syringed gently with normal salt solution or with mild antiseptic solutions (boric acid, liquor antisepticus alkalinus, etc.) repeated several times a day. Antiseptic gargles should be employed, or, in case the child cannot or will not use these, swabbing of the throat with cleansing and antiseptic solutions as for pharyngitis and tonsillitis of other nature. Diluted peroxide of hydrogen is often to be recommended, or solutions of potassium permanganate or of corrosive sublimate. When a pseudodiphtheritic membrane has formed application of diluted peroxide of hydrogen (1:2 of water) may be used, followed by diluted tincture of the chloride of iron (1:4) or of bichloride of mercury (1:5000), or nitrate of silver (gr. 5 or 10:oz. 1) (0.324 or 0.648:30). When local treatment is attended by great resistance on the part of the child, as is often the case, the advisability of continuing it is questionable. Its importance must be determined for each individual.

Otitis can probably be prevented to some extent by persistent disinfection of the throat and nose. The constant wearing of a flannel cap over the ears, and the use of a hot water bag or of hot water douching of the canal, is of benefit as a preventive or if the disease has developed. In the latter event paracentesis may become necessary. If perforation of the drum-head has taken place the usual treatment of suppurating otitis media is necessary.

Adenitis may often be aborted by the application of an ice-bag, tincture of iodine, flexible collodion, or a 15 per cent. ointment of ichthyol. The ice-bag should be of thin rubber and kept constantly in position, with a thin layer of cloth between it and the skin. If it is evident that suppuration will take place hot compresses or poultices may be employed to hasten the process and to relieve the pain. Early evacuation of the pus is indicated.

Nephritis requires prompt treatment. Whether anything can be done to prevent its development is a much disputed question. The avoidance of surface-chilling and the employment of an unirritating diet have already been mentioned. A salt-free diet has been urged by Pater,¹ Delearde² and others, but Nobécourt and Merklen³ found albuminuria less frequent when milk alone was employed. Widowitz,⁴ Buttersack⁵ and others have strongly recommended the administration of hexamethylenamine as a preventive measure, and Royer⁶ confirmed previously published reports upon the value of chloral given for the same purpose.

When nephritis has developed, if of but slight severity, the frequently repeated application of hot poultices over the renal region, the use of warm baths, and the administration of mild diuretics, such as acetate of potash, citrate of potash and sometimes of digitalis, are sufficient. Stimu-

¹ Bull. et. mem. soc. méd. des hôp., 1906, XXIII, 93.

² L'Echo méd. du nord., 1907, XI, 25.

³ Arch. de méd. des enf., 1908, XI, 81.

⁴ Wien. klin. Wochenschr., 1903, XVI, 1113.

⁵ Deut. Arch. klin. Med., 1904, LXXX, 356.

⁶ Penna. Med. Journ., 1907, X, 286.

lating diuretics must never be employed. When the nephritis is more severe, with anuria or great diminution of the amount of urine, convulsions, vomiting, and the symptoms of uremia, the treatment must be energetic. This is discussed more fully in the chapter upon Acute Nephritis, Vol. II, p. 181.

Arthritis may well be treated with salicylic acid combinations on the ground that it may be rheumatic in nature. If it is but slight, local protection of the joints may be all that is required. Purulent inflammation of the joints is to be subjected to surgical measures.

The treatment of other complications and sequels does not require special consideration here. During convalescence, especially from a severe attack, tonic measures are indicated, including the administration of iron to combat the anemia which often results.

CHAPTER III

MEASLES

(Rubeola. Morbilli)

Measles was undoubtedly known to the ancients, but was confounded by them with several other diseases. It was differentiated clearly from small-pox by Rhazes¹ about 900 A. D. and from scarlet fever by Sydenham² in the middle of the 17th Century.

Etiology. Predisposing Causes.—Race, sex, climate and locality, social position, and sanitation appear to exert no influence on the occurrence of the disease. The presence of other acute disorders occasionally delays the appearance of the symptoms of measles, but does not prevent it. *Age* is a very important factor. The susceptibility in the latter half of the 1st year is disputed, but it is agreed that infants in the first 6 months are much less often attacked than later. Yet even instances of congenital measles are recorded, Ballentyne³ having collected 21 such from medical literature. Additional cases of the presence of measles at or developing a few days after birth have been reported by Mason,⁴ Steinschneider⁵ and Rocaz.⁶

Whether the infrequency in nurslings is due to lesser susceptibility, or to lesser frequency of exposure is not certainly determined. Lichtenstein⁷ observed 40 nurslings attacked out of 47 exposed to infection, this appearing to be contrary to the usual experience. After the 1st year the influence of age ceases. That few exposed adults suffer from the disease clearly depends on the fact that so many have already had it. In the epidemic of 1846 in the Faroe Islands, described by Panum,⁸ where no one was thus protected, adults of every age proved as susceptible as children, and no instance was met with where an unprotected adult exposed did not take the disease.

¹ De variolis et morbillis, 1756.

² Processus integri.

³ Arch. of Pediat., 1893, X, 301.

⁴ Boston Med. and Surg. Journ., 1908, CLIX, 437.

⁵ Deutsch. med. Wochenschr., 1914, XL, 441.

⁶ Gaz. hebdom. des sci. mèd. de Bordeaux, 1906, XXVII, 260.

⁷ Hygeia, 1914, LXXVI, 1022. Ref. Monatsschr. f. Kinderh. Ref., 1915, XIV, 455.

⁸ Verhandl. d. physik.-med. Gesellsch. in Würzburg, 1851, II, 292, 293; Virchow's Archiv 1847, 1, 492.

The *individual susceptibility* is very great, exceeding that of most of the other infectious diseases. In the Faroe Islands' epidemic over 6000 of 7782 inhabitants were attacked, 1500 escaping only through the institution of absolute quarantine in certain villages. Later, in the epidemic of 1875, as described by Hoff,¹ 99 per cent. of the unprotected inhabitants of Thorshaven were affected. In my own experience measles is one of the most readily contracted of the diseases of its class, the susceptibility to it being so great that spread of the infection is little controllable. Yet a certain individual immunity, it is true, temporary or permanent, is occasionally met with. A decided *epidemic influence* exists also, measles being much more frequent or severe in certain years than in others, especially in localities where it is always endemic to some extent. This may depend upon a greater virulence of the poison in some years, and a consequent greater tendency to spread. There is no regularity in the return of these epidemic outbreaks. *Season* seems to possess a slight influence, the disease being more prevalent in winter, and especially in spring, than in summer. Of 530 epidemics studied by Hirsch² 339 (64 per cent.) occurred in the colder part of the year, and 191 (36 per cent.) in the warmer. In 213 of the epidemics the height was reached in 76 in spring, 59 in winter, 48 in autumn and 30 in summer. Yet the greater prevalence in the cooler months may depend upon the closer confinement and consequent more intimate association in houses and especially in schools.

Exciting Cause.—This is without doubt a germ of some sort. Canon and Pellicke³ and Czajkowski⁴ described a short bacillus constantly present in the blood and the nasal secretion. Bacilli have also been found in the blood by Arsamakov,⁵ Zlatogoroff,⁶ Von Niessen,⁷ Giarre and Picchi⁸ and others. Micrococci have been reported in the blood and the nasal mucus by Lesage⁹ and in the blood, throat, nose, and eyes by Tunnicliff.¹⁰ An ameba-like body was found in the red corpuscles by Doehle,¹¹ Behla,¹² and later by Rosenberger.¹³

In this connection the various experiments upon the possibility of the transmission of the disease to other animals are of interest and importance. Goldberger and Anderson¹⁴ succeeded in producing the disease in monkeys by inoculating with virus obtained from the secretions and the blood. As this was accomplished after passing the material through a Berkfeld filter, the etiological relationship of any of the microorganisms previously reported would appear to be disproved, and the germ must be ultra-microscopic. The transmissibility to monkeys has been confirmed by Hektoen and Eggers¹⁵ and by Lucas and Prizer.¹⁶ The true nature of

¹ Sundhedsskollegiets Aarsberetning 1876. Ref. von Jürgensen, Nothnagel's Encyclop. Amer. Ed., Measles, 228.

² Handb. d. histor. geog. Path. 1881, 116.

³ Berl. klin. Wochenschr., 1892, Apr. 317.

⁴ Centralbl. f. Bakt., 1895, XVIII, 517.

⁵ Bolnitch Gaz. Botk. 1898, 40. Ref. Rev. de méd., 1899, XIX, 561.

⁶ Centralbl. f. Bakt. 1904, XXXVII, 249.

⁷ Arch. f. Derm. u. Syph., 1902, LX, 429.

⁸ Acad. med. phys. florent., 1900.

⁹ Compt. rend. de la soc. de biol., 1900, LI, 1203.

¹⁰ Journ. Amer. Med. Assoc., 1907, LXVIII, 1028.

¹¹ Centralbl. f. allg. Path. u. path. Anat., 1892, III, 150.

¹² Centralbl. f. Bakt., 1896, XX, 561.

¹³ Amer. Med., 1906, XII, 139.

¹⁴ Journ. Amer. Med. Assoc., 1911, LVII, 476; 971; 1612.

¹⁵ Journ. Amer. Med. Assoc., 1911, LVII, 1883.

¹⁶ Journ. Med. Research, 1912, XXVI, 181.

the microorganism is, therefore, evidently not yet determined. It seems certain that under the influence of its presence, other germs, such as the streptococcus, pneumococcus and the bacillus of diphtheria, are enabled to produce complications.

The germ is very short-lived in measles as compared with scarlet fever and small-pox. Positive data are necessarily lacking, but it is probable that it dies in from 10 to 14 days from the onset of the attack. Its habitat is certainly the secretions of the mucous membrane of the nose, mouth, and eyes, and the blood; and it is probable that the desquamating epidermis has no infectious power unless contaminated by these. It was found without infectivity by Anderson and Goldberger. Inoculation experiments upon human beings, claimed to have been successful, were made with the blood by Home¹ and by Hektoen,² with the blood mixed with serum from the vesicles by Katona,³ and with the nasal mucus by Mayr.⁴ Some investigators have confirmed these results, while others have failed, and further studies on the subject are needed.

Method of Transmission.—This may be either mediate or direct, the latter occurring in the vast majority of instances. The germ is certainly very virulent, and but a brief exposure is necessary. The disease can be communicated by way of the air through the mere presence in the sick-room, but that it can be carried any distance by it is entirely unlikely. The experiments of Grancher⁵ and others indicate that it cannot. Many instances of transmission by fomites or by a third person are on record, but are certainly exceptional. The careful analysis made by Jürgensen⁶ throws doubt upon some of those oftenest quoted. Some more recent experiences in favor of mediate transmission have been published by Roch,⁷ Rohmer,⁸ Lanzarini⁹ and others. The common experience, however, agrees with that of Jürgensen. The usual absence of mediate transmission may be explained in part by the germ's short duration of life. Rooms which have been occupied by patients with measles can be used with safety by susceptible subjects very soon after the attack is over.

Period of Infectiousness.—It is probable that the danger exists during the last part of the stage of incubation, and certainly so from the onset of the invasion and during at least a portion of the period of eruption. The great frequency with which the disease is disseminated by schools and the difficulty of controlling its spread, proves the infectiousness of the early periods. The infectiousness lessens rapidly as the catarrhal symptoms disappear. Certainly the disease is communicated in but few instances during the stage of desquamation.

Pathological Anatomy.—There are few characteristic post-mortem lesions. According to Neumann¹⁰ the principal changes in the skin consist of dilatation of the vessels, with a surrounding infiltration of small cells, situated in the upper portion of the cutis and extending into the Malpighian layer, together with an extensive infiltration around the hair

¹ Medical Facts and Experiments, 1759.

² Journ. Infect. Dis., 1905, II, 238.

³ Oesterreich. med. Wochenschr., 1842, 697.

⁴ Zeitsch. d. k. k. Gesellsch. d. Aerzte zu Wien., 1852, VIII, 13.

⁵ Traité des mal. de l'enf., 2 Ed., I, 322.

⁶ Nothnagel's Encyclop. Amer. Edit., Measles, 247.

⁷ Arch. de méd. des enf., 1907, X, 292.

⁸ Jahrb. f. Kinderh., 1912, LXXV, 78.

⁹ La Pediat., 1907, XV, 366.

¹⁰ Med. Jahrb., 1882, 159.

follicles and the sudoriparous and sebaceous glands. Similar changes are seen in the mucous membrane, a catarrhal inflammation with a small-celled infiltration, occurring in the eyes and in the respiratory and alimentary tracts. The histology of the buccal eruption, as studied by Hlava¹ and by Flamini² consists in an interpapillary injection of the capillary vessels, an infiltration with round cells, thickening of the superficial layer of the epidermis and degenerative changes of the lower layer, forming a minute pustule.

The inflammatory process is followed by desquamation of the epithelium of both the skin and the mucous membrane. The lymphoid tissues throughout the body, including the lymphatic glands everywhere, the tonsils, spleen and the lymphatic follicles of the intestinal tract, exhibit decided cellular hyperplasia. Focal necrosis of the liver has been described (Freeman).³

Symptoms.—ORDINARY TYPE. **Period of Incubation.**—This period, up to the first appearance of catarrhal symptoms, has been very accurately determined by Panum⁴ and numerous other observers to be 10 or 11 days, or 14 days until the rash appears. Exceptionally it may be of slightly longer or shorter duration. In the majority of cases no symptoms occur. Meunier,⁵ however, insists that incubation is constantly attended by a very decided loss of weight beginning on the 4th or 5th day. The buccal eruption, presently to be described, is sometimes discovered before the catarrhal symptoms of the stage of invasion begin. Rolleston⁶ maintained that ephemeral prodromal eruptions, such as urticaria or erythema, appear during the last part of the incubative period in nearly half of the cases studied. The characteristic changes of the blood (p. 344) begin to show themselves during the incubative period.

Period of Invasion.—Occasionally a convulsion or decided chilliness ushers in the attack, or fever develops suddenly; but as a rule the onset is gradual, the symptoms being indistinguishable from those of a severe general cold. The child is irritable, tired, chilly, and often peculiarly drowsy. As the disease progresses there are decided lachrymation, photophobia, redness of the conjunctiva, coryza, sneezing, thirst, and often a dry, hard, and sometimes distressing cough. Occasionally a few dry râles are audible in the chest. Epistaxis is not uncommon. The upper lip is excoriated by the nasal secretion, the appetite diminished, and the tongue coated, with the edges perhaps somewhat red and the papillæ here a little enlarged. Some soreness of the throat is complained of, and the pharynx, fauces and tonsils are moderately congested, the latter being slightly swollen. Vomiting occasionally occurs, and diarrhea is not infrequent. Headache is common and delirium may occur.

By the 2d or 3d day the characteristic eruption may be seen on the mucous membrane (enanthem), consisting of small, red macules dotted over the hard and the soft palate and resembling those which appear later upon the skin. It is at its height when the cutaneous rash develops and may persist 3 or 4 days more. A condition of another sort, the so-called "buccal eruption" or "Koplik's spots," is found upon the mucous

¹ Časopis lékařů českých, 1906, 773. Ref. Zentralbl. f. inn. Med., 1906, XXVII, 923.

² Riv. di clin. pediat., 1908, VI, 401.

³ Arch. of Pediat., 1900, XVII, 81.

⁴ Loc. cit.

⁵ Gaz. hebdom., 1898, 1057.

⁶ Brit. Med. Journ., 1905, Feb. 4.

lining of the lips and cheeks, oftenest close to the junction of the latter with the upper jaw. It consists of minute bluish-white points each surrounded by a small slightly red areola. When the spots are numerous the areolæ coalesce, giving a redder tint to the whole lining of the cheeks and lips, over which are dotted the crowded spots.

Although the buccal eruption was previously described by Rinecker and by Reubold,¹ Flindt,² Monti,³ Filatow,⁴ and others, its diagnostic importance did not receive general attention until pointed out by Koplik⁵ (Fig. 63). It can be seen to advantage only in bright daylight, pref-

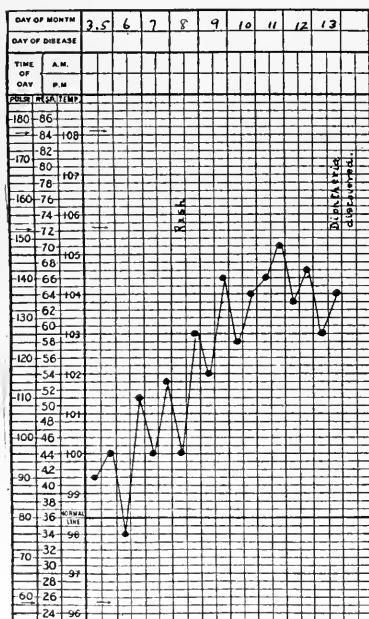


FIG. 64.

FIG. 64.—MEASLES WITHOUT PRE-ERUPTIVE FALL OF TEMPERATURE.

R. McN., 4 years old. Mar. 6, vomited; Mar. 8, well-marked catarrhal symptoms, buccal eruption, rash appearing on face and body; Mar. 10, attack severe; Mar. 13, diphtheria discovered.

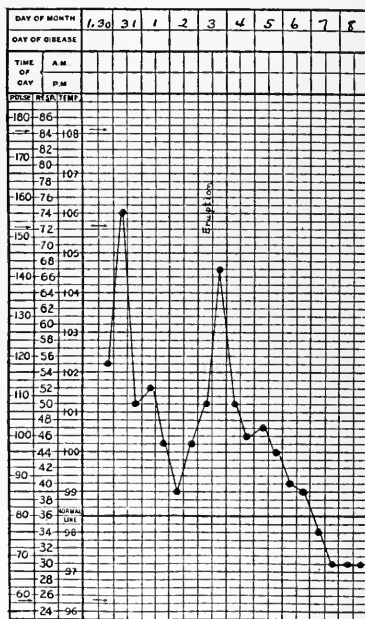


FIG. 65.

FIG. 65.—MEASLES WITH PRE-ERUPTIVE INTERMISSION IN TEMPERATURE.

Lena M. Jan. 30, catarrhal symptoms began; Feb. 2, symptoms been very pronounced, fall of temperature to 99°; Feb. 3, abundant eruption, general symptoms improved, but temperature elevated; Feb. 8, desquamation.

erably by carrying the child close to the window. Occasionally it may be found as much as 5 days before the cutaneous rash develops (Cotter)⁶ but is oftener seen 2 to 3 days before, and quite frequently is not discoverable until after this appears. The buccal eruption is almost pathognomonic of measles, and is present in from 80 to 90 per cent. of the cases. In the

¹ Virchow's Archiv., 1853, VII, 76.

² Ref. v. Jürgensen, Nothnagel's Encyclop., Amer. Ed., 1896, 286.

³ Jahrb. f. Kinderh., 1873, VI, 20.

⁴ Dis. of Child. Amer. Transl., 1904, I, 97; II, 660.

⁵ Arch. of Pediat., 1896, XIII, 918; Med. News, 1899, LXXIV, 673.

⁶ Arch. of Pediat., 1906, XXVII, 923.



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

FIG. 63.—THE PATHOGNOMONIC SIGN OF MEASLES (KOPLIK'S SPOTS).

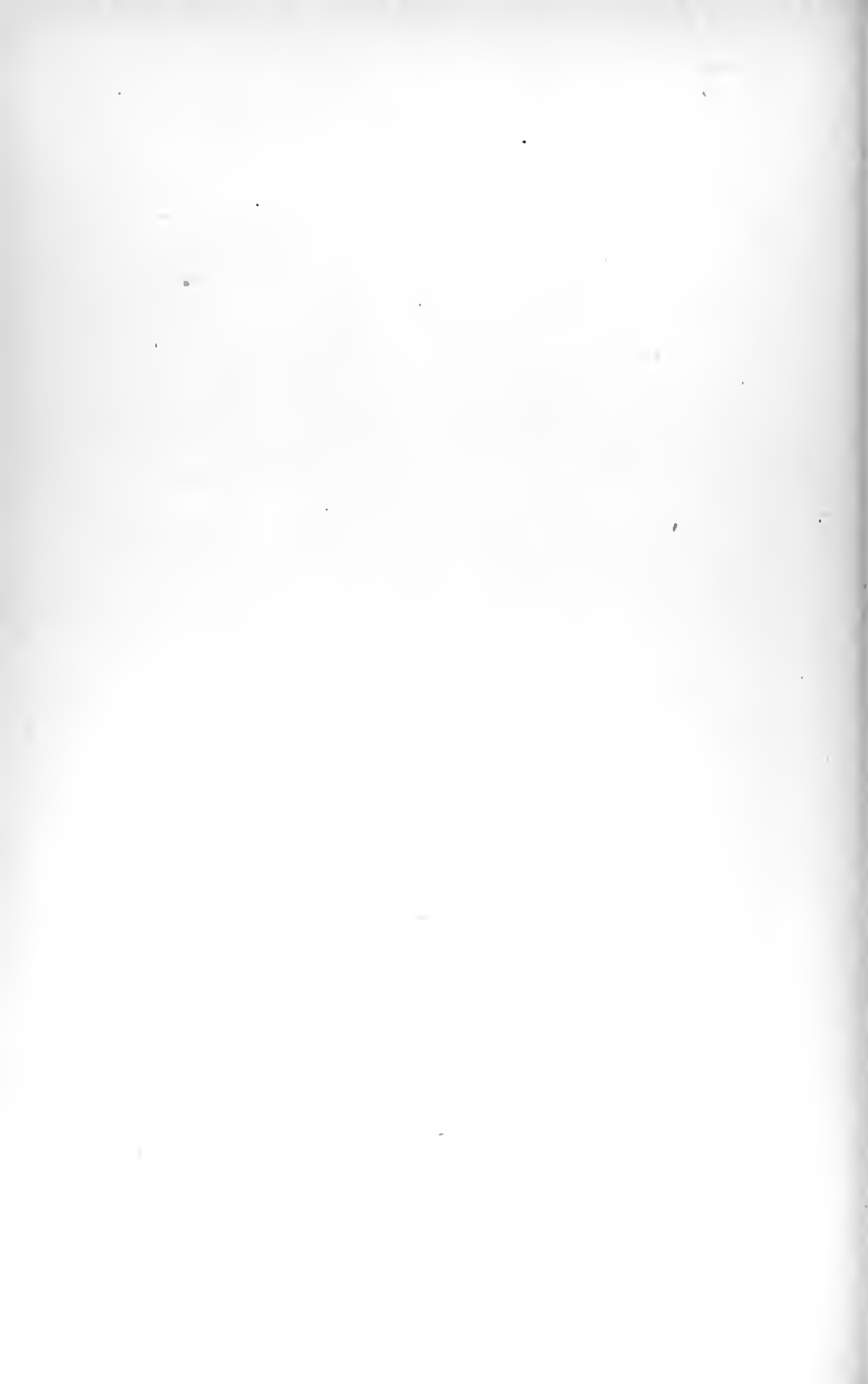
FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in line. Minute *yellow points* are surrounded by a red area. Always discrete.

(*Medical News*, 1899, lxxiv, 673.)



remaining, repeated, careful inspection will fail to reveal it. It disappears by the time, or even before, the cutaneous eruption is fully developed.

Prodromal cutaneous eruptions are not infrequently observed in this stage, if not seen earlier. Thus from a number of hours up to a day or more before the characteristic eruption is distinctly visible, there can sometimes be observed an alteration in the appearance of the skin of the face, suggesting a roughness, reddening or mottling situated, as it were, *beneath* the surface. In other cases there is a distinct localized blotchy erythema or an urticaria, or a more or less widely diffused scarlatiniform rash, or even a faint eruption which suggests the beginning of the ordinary rash. These prodromal eruptions are, in my experience, common. They usually disappear before the true exanthem of the disease becomes manifest.

All the catarrhal symptoms are well marked by the 2d day of invasion, and increase steadily in severity through this stage. The temperature curve during this period is subject to variations. Very frequently it increases steadily to 102° to 104°F. (38.9° to 40°C.), with ordinary morning remissions (Fig. 64). In very many cases, however, after a sharp initial rise there occurs about the 2d or 3d day a decided remission or even intermission of both evening and morning temperature, followed by the redevelopment of fever before the eruption appears (Fig. 65). Occasionally the cutaneous eruption begins to develop before the temperature rises again (Fig. 66). This fall of temperature is entirely unattended by any amelioration in the other symptoms. It has been considered by many writers to be characteristic of the disease. Others maintain that the curve without intermission occurs as frequently as the other, and this has been my own experience. Bolognini¹ describes a very slight sensation of crepitation obtained by palpation of the abdomen during the stage of invasion. His observations are confirmed by Koppen.² The duration of the stage of invasion in typical cases is 3 or 4 days. Irregularity in its duration and course will be referred to later (p. 345).

Period of Eruption.—The Rash.—The characteristic rash is found on the skin by the end of the 3d or the morning of the 4th day of the disease in ordinary cases; sometimes not until the 5th day. It develops first either upon the forehead, scalp, cheeks, temples, behind the ears, or about

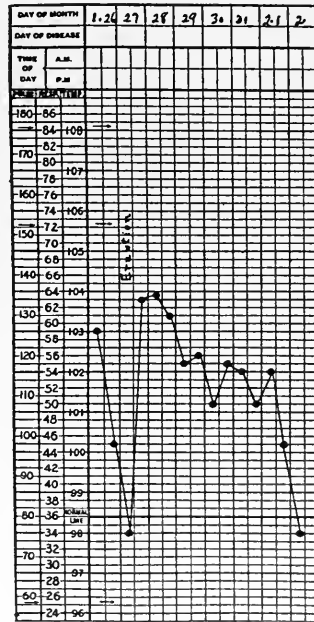


FIG. 66.—MEASLES WITH PRE-ERUPTIVE FALL OF TEMPERATURE CONTINUING WHEN RASH FIRST APPEARED.

Howard M., aged 7 years. Jan. 25, catarrhal symptoms and fever; Jan. 26, temperature falling; Jan. 27, abundant eruption on face, spreading to body; temperature still afebrile; Jan. 30, rash almost gone, temperature still elevated; Jan. 31, desquamation.

¹ La Pediat., 1895, III, 110.

² Centralbl. f. inn. Med., 1898, XIX, 673.

the mouth; the exact situation probably varying with the case. It then spreads with variable rapidity, but as a rule is not only more abundant by its 2d day in the regions where first seen, but has extended over the whole body, possibly excepting the legs and forearms, feet and hands, which may not be involved until its 3d day. Even the palms and soles finally exhibit it. The *individual spots* are at first of small pin-head size, pale-red, not elevated, round or irregular in shape, and discrete with the

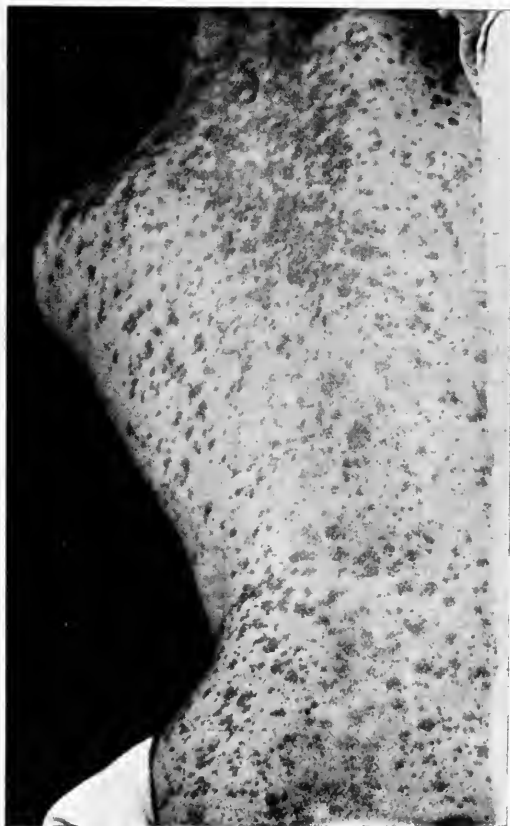


FIG. 67.—ERUPTION OF MEASLES.

Boy of 12 years, showing the characteristic grouping exhibited by the eruption.

surrounding skin healthy. They rapidly enlarge up to the size of a large split pea, become distinctly elevated to sight and touch and of a darker red color, commonly with a slightly bluish cast. There exists a decided tendency to grouping, a series of spots becoming confluent by their margins into irregular, short, straight or curved lines—the well-known “crescentic eruption” (Fig. 67). These groupings are separated by small white channels of healthy skin, the contrast producing the appearance characteristic of this disease. In some portions of the body, especially the face, back, buttocks and the inner surface of the thighs, the rash may become confluent in large areas, the intervening channels disap-

pearing to a large extent. Even here, however, the peculiar uneven roughness and lack of uniformity in coloring are quite evident.

The individual spots reach their fullest development in about 24 hours, and then begin to fade. Pressure by the finger will at first completely obliterate them for a moment; later a slight discoloration remains. Taken as a whole the rash is at its height on the 2d or 3d day of the eruptive period and then fades, beginning in the situation where it first appeared. It may consequently be diminishing on the face while not yet at its height upon the legs. All traces of the eruption have disappeared by the

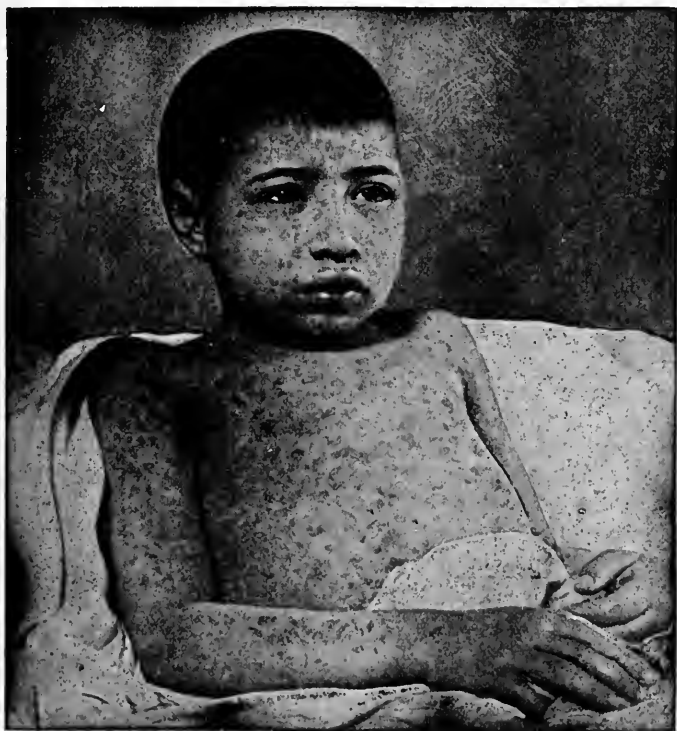


FIG. 68.—FACIES IN MEASLES.

Girl with measles, showing the peculiar heavy and swollen appearance of the face.

4th or 5th day after the first appearance, except a pale-yellowish or brownish pigmentation which very commonly persists for a decidedly longer time.

Other Symptoms of the Eruptive Stage.—The fever generally increases with the development of the rash and reaches its maximum with this upon the 2d or 3d day of the eruptive stage, with only slight morning remissions. There are, however, very many exceptions to this rule, and it is of frequent occurrence for the temperature to fall almost by crisis even upon the 2d day of this stage, while the rash is still at its height, or even before it has attained this. All the catarrhal symptoms meanwhile persist or increase in severity as the rash develops. On the 2d day of the rash there is present a very decided puffiness of the face, which, with the photophobia, conjunctivitis, severe coryza, obstructed

nasal respiration, and excoriation of the upper lip give to the child a very characteristically stupid expression (Fig. 68). The eyes secrete freely, and the edges of the lids stick together during sleep. The voice is hoarse, cough is often distressing, and numerous râles can generally be heard in the chest. The tongue is coated, and in severe attacks may become dry or even denuded. The inflammation of the fauces and pharynx continues but is seldom very severe. Thirst is great, the appetite is lost, and diarrhea is a frequent and sometimes troublesome symptom. Vomiting is not common. There may be slight delirium during the height of the attack, or the drowsiness of the prodromal stage may persist. The superficial lymphatic glands throughout the body are swollen and often tender. As I have pointed out elsewhere¹ this enlargement is quite commonly so considerable that its well-recognized occurrence in rubella as well cannot be considered so diagnostic a symptom of the latter disease as is often supposed. There is often very annoying itching of the skin. Febrile albuminuria sometimes occurs, there is a marked diazo-reaction, diacetic acid and propeptone may sometimes be found in the urine, and a moderate urobilinuria is present (Rach and Reuss).²

The blood was studied by Renaud³ and since then by Flesch and Schossberger,⁴ Hecker,⁵ Lucas⁶ and others. There may be a transient lymphocytosis early in the incubation, but from 2 to 6 days before the first symptoms appear there occurs a leucopenia with a very characteristic relative diminution or even disappearance of the lymphocytes, the blood-picture showing a predominance of polymorphonuclear cells. This diminution of the lymphocytes shows itself several days before the development of the buccal eruption. During invasion the number of polymorphonuclear cells becomes diminished in uncomplicated cases both relatively and absolutely, and the blood-picture again becomes lymphocytic. A hypoleucocytosis, especially of the polymorphonuclear cells with increase of the mononuclear cells is very decided in, and quite characteristic of, the early eruptive stage. The eosinophiles are diminished in number. The normal number of leucocytes is soon attained as convalescence advances. Hyperleucocytosis develops if complications are present.

When the rash begins to fade on the 2d or 3d day of the eruptive period, the temperature, if still elevated, falls rapidly by lysis, often reaching normal in 1 or 2 days after the fall begins. The catarrhal symptoms also improve rapidly, keeping pace with the temperature, although hoarseness, cough, and irritation of the eyes frequently last for several days longer. The average duration of fever is about 7 days in all, but often less. (See Figs. 65 and 66.) The frequency of respiration and pulse is in proportion to the elevation of temperature, unless complications appear. The rapidity of the disappearance of the symptoms is generally very striking.

Period of Desquamation.—The eruptive and desquamative stages cannot be very sharply demarcated. As a rule, the stage of desquamation may be said to begin with the disappearance of the eruption, not including the pigmentation, and with the subsidence of other symptoms on

¹ Univ. Med. Mag., 1892, June.

² Zeitschr. f. Kinderh., Orig., 1911, II, 460.

³ Thèse de Lausanne, 1900. Ref. v. Jürgensen. Nothnagel's Encyclop. Amer. Med. Measles, 335.

⁴ Jahrb. f. Kinderh., 1906, LXIV, 724.

⁵ Zeitschr. f. Kinderh., Orig., 1911, II, 77.

⁶ Amer. Journ. Dis. Child., 1914, VII, 149.

the 7th or 8th day of the disease, although the actual scaling may not appear until 1 or several days later. The desquamation consists of very fine, branny scales. As a rule it is found first on the face and later elsewhere, following the order in which the eruption made its appearance. It continues a few days to a week, or occasionally longer. The amount of desquamation is usually in proportion to the intensity of the eruption; but although sometimes extensive and very noticeable, it is generally slight and most evident on the face, and in many cases cannot be detected at all. There are no symptoms characteristic of this stage, except in some cases a persistence of the catarrhal manifestations, especially the cough and the conjunctival irritation.

VARIATIONS FROM THE ORDINARY TYPE.—Many variations from the type described may mark the attack either as a whole or in certain particulars.

The *duration of incubation* may be altered, but a stage of less than 8 days or more than 12 days is unusual. As short a duration as 5 days and as long as 3 weeks are on record. Occasionally symptoms are observed consisting of loss of appetite, slight fever, malaise, and perhaps slight indications of the catarrhal condition to follow. Exceptionally other symptoms appear during this period, but such are to be regarded as accidental or anomalous.

The *stage of invasion* may be unusually protracted, lasting perhaps 6 to 8 days. Barthez¹ reports an instance of invasion continuing 16 days. It is, however, more prone to be abnormally short, lasting only 1 to 2 days, and sometimes there appears to be no period of invasion whatever. I have seen entire local epidemics of measles in institutions characterized by an absence or very slight development of prodromer. The combined length of incubation and invasion is fairly constantly 14 days. If, then, one of these periods is lengthened or shortened, the other must vary inversely with it. There are, of course, exceptions in which the total duration of the two periods is shorter or longer than the figure given. In other cases the initial symptoms may be unusually intense; stupor, convulsions, continuously high fever and severe diarrhea or vomiting being among these. The respiratory symptoms may be excessive, the secretion from the eyes and nose being profuse, and the breathing difficult, or croup may complicate the condition. Violent epistaxis may occur, or other complications may influence the character of this stage. In one instance under my own observation a well-marked lobar pneumonia represented the chief of the symptoms of invasion. This was followed at the proper time by the development of the eruption and of other evidences of measles, while the pneumonia meanwhile underwent rapid resolution. Bronchopneumonia is also reported as a prodromal symptom. These unusually severe initial symptoms may continue into the eruptive stage, or may ameliorate when the eruption appears.

The *eruptive stage* is also subject to numerous variations, giving distinct types to the attack. First is to be mentioned the mild form. In this the child is so little ill that it is not confined to bed, and suffers practically no discomfort. After a short invasion the catarrhal symptoms continue to be of very mild character and the fever is only slight, or even entirely absent (*Rubeola afebrilis*) (Fig. 69). The eruption is often pale-

¹ Barthez and Sanné, *Malad. des enf.*, 1891, III, 17.

red, scanty and very little confluent, strongly suggesting rubella, or so poorly developed that diagnosis is very difficult.

An **abortive form** is characterized by a well-marked stage of invasion, but with an eruptive stage which, sometimes typical at the onset, sometimes poorly developed, rapidly disappears together with the attending symptoms (Fig. 70). **Measles without eruption** (*Rubeola sine eruptione*)

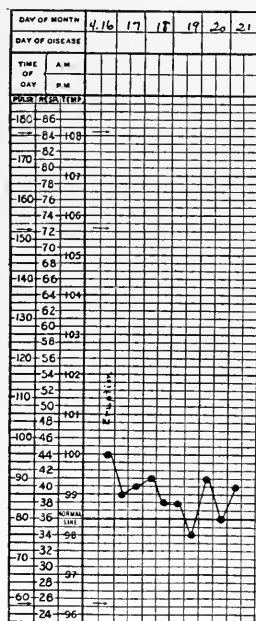


FIG. 69.

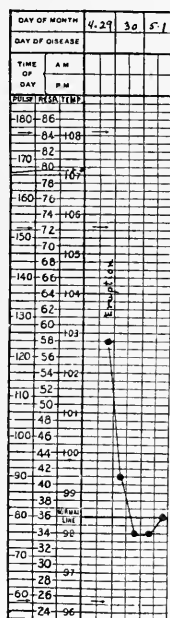


FIG. 70.

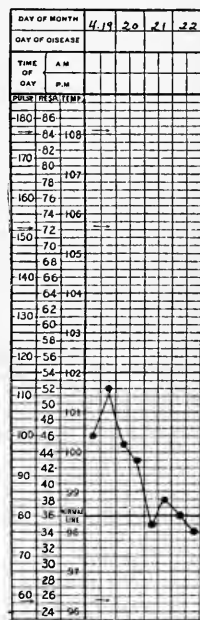


FIG. 71.

FIG. 69.—RUBEOLA AFEBRILIS.

Richard G., aged 8 years. Apr. 15, vomited, lachrymation; Apr. 16, vomited again, characteristic rash after a mustard-bath, mild catarrhal symptoms throughout attack, no fever during eruptive period, and no indication of it earlier.

FIG. 70.—ABORTIVE MEASLES.

Frank A., aged 4 years. Apr. 29, for 3 days had coughing, sneezing and drowsiness, typical eruption appeared today on face, catarrhal symptoms continue; Apr. 30, rash spread yesterday, now fading; May 1, rash nearly disappeared.

FIG. 71.—RUBEOLA SINE CATARRHO.

Willie B., aged 10 years. Apr. 18, no symptoms noted; Apr. 19, no catarrhal symptoms except a very slight redness of the eyes, eruption developing; Apr. 20, eruption more abundant, no catarrhal symptoms whatever; Apr. 21, rash fading. House epidemic.

has been described, catarrhal symptoms being present unattended by rash. Embden¹ reports 20 instances in one epidemic of 461 cases. So, too, cases are observed in which the characteristic rash occurs but without catarrhal symptoms (*Rubeola sine catarrho*) (Fig. 71). It is almost certain that the great majority of the cases apparently of this variety are instances of rubella or some disorder of the skin. Only in house epidemics could the diagnosis of either of these two forms be made, and then only with great reserve.

¹ Inaug. Dissertat. Heidelberg, 1889.

The *severe forms* of measles, apart from the influence of complications, may be of several varieties, although no sharp line of distinction separates one from another. In one, which may be called the **prolonged type**, the fever lasts an unusually long time. This, as a rule, depends upon the persistence of catarrhal symptoms, and especially upon a more than ordinary degree of bronchitis. In other severe cases the virulence of the poison is very great. All the symptoms exhibit an unusual severity from the outset, the eruption being very intense, widespread, confluent, and of a dusky, blue-red color. In other instances the disease begins

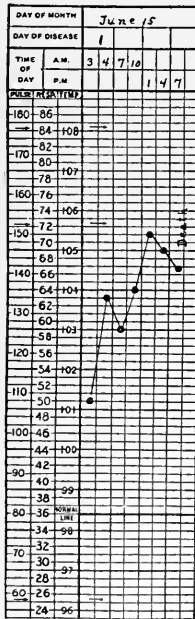


FIG. 72.

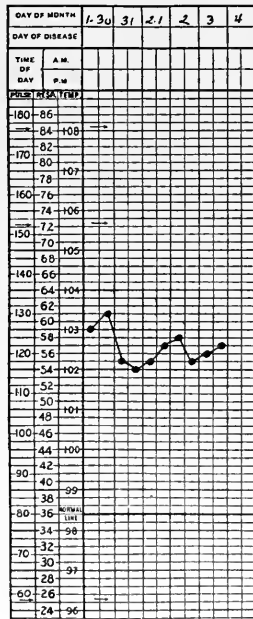


FIG. 73.

FIG. 72.—MALIGNANT MEASLES.

Robert McD., aged 16 months. June 14, vomiting, purging, fever, convulsions, said to have had fever several days, older brother convalescent from measles; June 15, faint macular rash after hot bath, convulsions and unconsciousness continued, hydrocephalic cry, death in evening of the 1st day of the eruption.

FIG. 73.—HEMORRHAGIC MEASLES.

Willie P., aged 5 years. Jan. 30, catarrhal symptoms noted; Jan. 31, severe chill, cyanosis, unconsciousness, well-developed rash followed hot bath; Feb. 3, been growing steadily worse, tongue dry, delirium; Feb. 4, eyes sunken, crowded petechiae on abdomen, larger purpuric patches on legs. Death.

in the ordinary way, but soon assumes a severe type. In still others some of the symptoms are but little marked. This is especially true of the rash, which may be poorly developed throughout the attack and frequently late in appearing, or which often shows a remarkable tendency to repeated disappearance, even when brought temporarily into evidence by hot baths ("Retraction of the eruption"). It was long erroneously supposed that this "striking in of the rash" was the cause of the unfavorable symptoms. It is, however, only an evidence of the feebleness of the circulation. Many of these cases with retraction

assume what has been called the **typhoid form**. The rash is then at no time well developed, there is great prostration, dry tongue, rapid and weak pulse, rapid respiration, high temperature, low delirium, restlessness, twitching, and perhaps repeated convulsions.

The most severe type of the disease is the **malignant form**. The symptoms are even more severe than those described, the course of the disease rapid, and death may take place before the time is reached for the eruption to appear (Fig. 72). The temperature in some of these severe cases may reach 108° to 109°F. (62.2° to 62.7°C.). The **hemorrhagic type**, or "black measles" of the older writers, is another severe type, really a malignant variety, less often seen than formerly. It occurs only in subjects already much debilitated. The rash is pale and never well developed. Hemorrhages take place into the spots and elsewhere into the skin as well as into the muscles and from the mucous membrane. There is delirium and great prostration (Fig. 73).

There are a number of **minor variations** from the ordinary type depending upon *characteristics of the eruption*, none of them possessing any special significance. Sometimes the rash appears upon the trunk before it does upon the face. Infrequently the macules are very small and so closely placed that the rash of scarlet fever is simulated. In some mild cases the rash appears only upon some one part of the body, especially the face and neck. In *Morbilli papulosi* the eruption consists of large, deep-red spots more papular than usual. *Morbilli miliares* exhibits minute vesicles upon the usual eruption. A *Morbilli bullosi* has also been described, although rarely seen, an eruption resembling pemphigus replacing or being combined with the ordinary rash of measles. The term, *Ecchymotic measles* is well applied to the cases so frequently seen in which extravasation of blood or of blood-coloring matter has taken place into the rash, especially on the extremities. The deep-red-dish-purple markings thus produced are uninfluenced by pressure, and remain distinct for days or even weeks after all other symptoms of the disease, except desquamation, have disappeared. To call this form "hemorrhagic measles" as is sometimes done, is a source of confusion, since there are none of the grave symptoms present which characterize the latter affection.

Complications and Sequels.—The younger the child the more frequent and more serious are the complications and sequels liable to be. After early childhood they are comparatively uncommon. Most important and oftenest seen are those connected with the *respiratory tract*. Exceptionally epistaxis may be so severe or so often repeated that it becomes an important complication. Chronic nasal discharge may be a sequel. Catarrhal laryngitis is always present to some extent and may at times be attended by so much mucous secretion, spasm or edema that severe laryngeal stenosis develops and even death may occur. When the stenosis depends on the first two factors it is intermittent; when on the last it is more or less constantly present. Stenosis is always a dangerous complication unless of very short duration. Ulcerative laryngitis is a not uncommon complication or sequel. Membranous laryngitis may be due to the presence of either a streptococcus or of the diphtheria bacillus. (See Vol. II, pp. 21 and 449.) The first occurs oftenest early in the attack, the second at a later period. The symptoms are much the same and a positive diagnosis can be made only by bacteriological examination. Gannelon¹ found membranous laryngitis in 14.4 per cent. of 1633 cases of measles.

¹ La rougeole a l'hospice des enfants assistées, 1892.

A subglottic laryngitis may occur and cause great disturbance of respiration, and even death, no membrane being discoverable.

Trachitis and bronchitis belong to the regular symptoms of the disease. The latter may sometimes become so severe that it prolongs the fever decidedly and may then be looked upon as a sequel.

Bronchopneumonia, frequently tuberculous, is very common especially in infancy and early childhood and is always most serious. It may develop at any time in the course of the disease, but oftenest during the eruptive stage or as a sequel after a short intermission. The statistics regarding the frequency of the occurrence of bronchopneumonia vary considerably. Jürgensen¹ found them ranging from 6 per cent. to 16 per cent. of the cases of measles.

Croupous pneumonia is a much less common complication, but is occasionally seen. Gangrene of the lung and pleurisy with effusion are observed exceptionally and bronchiectasis has been recorded as a sequel.

The *gastroenteric tract* likewise furnishes many complications and sequels. Some degree of catarrhal stomatitis is one of the regular symptoms of the disease. Aphthous and ulcerative stomatitis are frequently seen. Gangrenous stomatitis is rare, yet measles perhaps more than any other disease predisposes to it. Membranous pharyngitis is not uncommon, patches developing on the tonsils and adjacent parts. Like membranous laryngitis, with which it is often combined, it may be streptococcic or truly diphtheritic in nature. Diarrhea is not infrequently sufficiently severe to constitute a complication. Oftener it continues as a sequel, sometimes in a very chronic form. It may be simply catarrhal in nature, or may depend upon ileocolitis. This latter is especially liable to develop in summer time in debilitated subjects under 2 years of age. Le Lyonais² collected 18 instances of appendicitis occurring as a sequel to measles.

Otitis is of frequent occurrence, varying with the epidemic. In a mild non-suppurative form, it is very common in the early part of the eruptive stage. In a more severe form, with pain, suppuration and fever, it occurs oftener as a sequel about the end of the 2d week. Both ears are then usually affected, permanent injury may result, or meningitis may follow. Of 501 cases of chronic disease of the middle ear published by Downie³ 26.1 per cent. owed their origin to measles. Yet as a rule chronic otitis follows much less often than after scarlet fever.

Inflammation of the eyes of various sorts is often observed with or after the attack. Catarrhal conjunctivitis is one of the symptoms of the disease and can be considered a complication only when unusually severe. It is frequently very persistent in poorly nourished children. Keratitis and iritis may occur as sequels and optic neuritis is occasionally seen, Griscom⁴ having collected 23 cases in addition to 1 reported by him.

Circulatory affections are uncommon. Endocarditis, pericarditis and myocarditis are rarely seen. Thrombosis of the vessels in different parts of the body may exceptionally occur (Leitz).⁵ In the extremities this may produce gangrene of the limb. *Swelling of the thyroid gland* has been reported. Of *genito-urinary* complications nephritis is generally considered unusual. I have, however, seen it not infrequently either with

¹ Nothnagel's Encyclop. Amer. Ed., 318.

² Thèse de Paris, 1913-14, No. 109. Ref. Brit. Jour. Child. Dis., 1914, XI, 234.

³ Brit. Med. Journ., 1894, II, 1163.

⁴ Ann. of Ophthalm., 1912, XXI, 42.

⁵ Berl. klin. Woch., 1913, I, 1566.

the attack or later. Acute degenerative lesions of the kidney develop in malignant cases. Pyelitis may occur. Ulcerous vulvitis and gangrene of the vulva are very rare sequels, to which, however, measles especially predisposes.

Affections of the bones and skin have been reported as complications, among them gangrene, herpes zoster, herpes labialis, urticaria, erythema, and furunculosis. Osteomyelitis and necrosis have been recorded. Arthritis is a very uncommon sequel. Generalized cutaneous emphysema has been reported.

Nervous affections are unusual. Meningitis occurs occasionally as a sequel to otitis, or dependent upon a complicating tuberculosis. A dull apathetic condition is exceptionally seen, lasting for weeks after the disease is over. I have observed it in 1 instance. In 2000 cases of imbecility Beach¹ found 11 which dated from an attack of measles. Chorea and epilepsy have been reported as sequels and tetany has been occasionally observed during the attack. Convulsions sometimes develop during the attack and then constitute a very unfavorable indication. Paralysis of various forms may follow measles. It may be cerebral, myelopathic or peripheral in nature. The subject has been reviewed by Allyn,² Brückner,³ and others.

Other *infectious diseases* may exist in conjunction with measles, or as sequels or predecessors to it. One of the most common and unfavorable combinations is that of measles and diphtheria, the latter being prone to develop during the convalescent stage of the former. In 3400 cases in the Medical Asylums Board's Hospitals (Rolleston)⁴ 2.10 per cent. developed diphtheria. Scarlet fever and measles often occur together, or one immediately after the other. The combination of varicella, erysipelas, vaccinia, grippe, or typhoid fever with measles is occasionally seen, or the immediate following or preceding of the latter by one of the others. Epidemics of pertussis are particularly liable to precede or follow or to prevail at the same time with epidemics of measles. Measles, too, is certainly very prone to be followed closely by evidences of tuberculosis or, if the latter disease has already manifested itself, to increase its further development. Very many of the cases of bronchopneumonia attending measles are tuberculous in nature, and osseous and glandular tuberculosis are frequent sequels. For some reason there is often a temporary insusceptibility to the tuberculin reaction during the occurrence of an attack of measles.

Relapse.—This is encountered usually with great rarity, although Leade⁵ observed it 4 times in an epidemic of 262 cases. Undoubtedly many reported instances are errors in diagnosis. It takes place in from the 2d to the 4th week or sometimes later, after the attack is apparently over but while the original infection is still present in the system. It consists in the return of some or all of the characteristic symptoms of the disease. The mere re-development of some one symptom during the attack, as, for instance, the reappearance of the rash, does not constitute a relapse. As a rule the relapse is less severe and of shorter duration than the first attack.

Recurrence.—In spite of the widespread belief among the laity to the contrary, second attacks of measles are of very great rarity. This

¹ Brit. Med. Journ., 1895, II, 707.

² Med. News, 1891, LIX, 617.

³ Jahrb. f. Kinderheilk., 1902, LVI, 725.

⁴ Brit. Jour. Child. Dis., 1915, XII, 21.

⁵ Lancet, 1905, II, 1837.

is the opinion of the majority of authorities. Most instances of so-called recurrence are instances of errors in diagnosis, which circumstances make particularly easy in this disease. Maiselis¹ collected only 21 cases from medical literature, while he found 154 of scarlet fever. In 1100 cases of the disease Widowitz² observed no instance of a second attack.

Prognosis.—The prognosis of measles is generally good, the mortality averaging from 3 to 6 per cent., often less, but sometimes reaching much higher figures. During 5 years there occurred in Philadelphia 50,715 cases of measles, with a mortality of 1.6 per cent. (Graham).³ The number of cases of measles is, however, so great that the number of deaths from it is large. Comby⁴ gives 20,518 fatal cases in Paris during 19 years and McCollum⁵ states that the deaths during 5 years in London per 10,000 of the population were 3 or 4 times more numerous from measles than from scarlet fever. The greatest number of fatalities is observed in the 2d week of the attack.

Many factors influence the mortality. That of the *epidemic* is one of the most noteworthy of these, the disease being much more fatal in some years than in others. At times under unfavorable conditions the death-rate has exceeded 30 per cent. of the cases, while at other times it does not reach 2 per cent. In 1914 in 170,004 cases of measles occurring in portions of the United States the mortality was 1.73 per cent. (Wilson).⁶ Age, also, exercises a powerful influence. In general the younger the patient attacked during infancy and childhood the graver the prognosis, and the number of fatal cases is very much diminished after the 5th year. The disease appears to be milder and the mortality less in the first 6 months than in the succeeding months of the 1st year. Variot⁷ found a mortality of 12.31 per cent. among 601 cases of the disease in children. In the 1st year the mortality was 32.72 per cent. and in the 2d year 29 per cent. The greatest actual number of deaths occurred in the 2d year, there being fewer cases of the disease in the 1st. Of 367,602 deaths from measles reported in England and Wales during 40 years 335,874 were in children under 5 years of age (Williams).⁸ Henoch's⁹ statistics for the Charité Hospital gave a mortality of 55.6 per cent. in the first 2 years of life and 9.3 per cent. for from 3 to 11 years. In some epidemics, however, the mortality has been notably high among adults. This was the case in the Faroes' epidemic of 1845 (Panum¹⁰), and Kilbourne¹¹ reported a severe epidemic of 600 cases occurring in barracks with a mortality of 5 per cent.

Debilitating influences, such as want, exposure, crowding, imperfect hygiene in general, and neglect of treatment, increase the death-rate greatly. It is probably such factors which have caused the high mortality in foundling asylums and other institutions for infants and children, and among soldiers in camps. The proportion of fatal cases among the poor in institutions is always much higher than in private practice among the better classes. Of 1575 children treated during 5 years in the Hospice

¹ Virchow's Archiv, 1894, CXXXVII, 468.

² Wien. klin. Wochenschr., 1909, XXII, 1596.

³ Jour. Amer. Med. Assoc., 1917, LXVII, 1272.

⁴ Traité des mal. de l'enf., 2d ed., III 347.

⁵ Bost. Med. and Surg. Journ., 1903, CXLVIII, 31.

⁶ Arch. of Pediat., 1916, XXXIII, 261.

⁷ Bull. de la soc. de pédiat., 1904, No. 1.

⁸ 20th Cent. Pract. of Med., XIV, 120.

⁹ Ref. Williams, *loc. cit.*, 121.

¹⁰ *Loc. cit.*, 292.

¹¹ Mil. Surgeon, 1912, XXXI, 294.

des Enfants-Assistés in Paris, 728 died; *i.e.* 46.22 per cent (Comby).¹ Rolleston² reported a mortality of 12.6 per cent. in 3400 cases in the Metropolitan Asylums Board's Hospitals in 1913, most of the children being of the poorer classes.

Complications and sequels are far oftener the cause of death than is the disease itself. So, too, the occurrence of measles as a secondary affection to other diseases adds greatly to the danger. Bronchopneumonia, tuberculous or otherwise, perhaps occasions more deaths than any other complicating affection, but diphtheria and diarrhea are the factors in many instances. Of 157 cases of the combination of measles and diphtheria published by Blakely and Burrows³ 34 per cent. died. Even after the disease seems completely over, death often results from the development of fatal sequels, especially tuberculosis.

Certain *unfavorable symptoms* may be referred to. A high temperature during invasion indicates that the attack will probably be a severe one. A poorly developed eruption or one which retrocedes readily, combined with marked general symptoms, is of unfavorable import. High fever persisting while the other symptoms are disappearing suggests the presence of some complication. The development of unusual hoarseness may denote the existence of severe laryngitis.

Diagnosis.—The diagnosis of measles, although generally easy in the eruptive period, is sometimes attended by great difficulty. It rests principally upon the long prodromal stage with the attending fever and catarrhal symptoms, and the development later of the characteristic eruption. In the stage of invasion the resemblance of the catarrhal symptoms to those of a severe cold is very close. The fever is perhaps unduly high for the latter condition, and the symptoms in general too severe; yet positive diagnosis at this stage would be impossible were it not for the presence of the spots upon the palate, and especially of the characteristic buccal eruption. Very exceptionally the latter has been reported present in pertussis and follicular tonsillitis (Michelazzi)⁴ and in rubella (Widowitz;⁵ Müller).⁶

Yet since few observers claim to have seen it in any other disease than measles, the presence of the buccal eruption is a very valuable diagnostic sign. Its absence, however, is not proof that the disease is not measles. The diminution of the lymphocytes is of diagnostic value during incubation, the relative increase of polymorphonuclear cells then distinguishing measles from the first stage of pertussis, in which there is an increase of the lymphocytes (Renaud).⁷ The neutrophilic hypoleucocytosis and the diminution of the number of the eosinophiles are a diagnostic aid in the eruptive stage, being exactly the opposite of the condition seen in scarlet fever.

Rubella is the disease which resembles measles most closely. The chief points of distinction are the shortness and mildness or absence of prodromes in rubella; the slight degree of catarrhal symptoms; and the more rapid development of the eruption, its absence of grouping, and its more multiform, paler and fugacious character. The degree of glandular enlargement is of little aid in diagnosis. (See Rubella.) Yet

¹ *Traité des malad. de l'enf.*, 2d ed., III, 348.

² *Brit. Jour. Child. Dis.*, 1915, XII, 129.

³ *Bost. Med. and Surg. Journ.*, 1901, CXLV, 89.

⁴ *Gaz. degli. osp. a delle clin.*, 1904, XXV, 35.

⁵ *Wien. klin. Wochenschr.*, 1899, XII, 919.

⁶ *Münch. med. Wochenschr.*, 1904, LI, 98.

⁷ *Thèse de Lausanne*, 1900.

in some atypical cases of rubella the rash is characteristically morbilliform in character, and the catarrhal symptoms are marked, while in some instances of measles the symptoms, including the eruption, are poorly developed and strongly suggest rubella. In sporadic cases of measles the diagnosis is consequently often impossible unless the buccal eruption is discovered.

Scarlet fever can cause difficulty only in atypical cases. Such cases sometimes exhibit a very blotchy rash somewhat resembling measles, while, on the other hand, the rash of measles may at times be so confluent that scarlet fever is suggested. Careful examination will, however, show decided differences in the two eruptions. That of measles is always slightly uneven to the touch and areas or channels of healthy white skin can always be found in some localities. The chin and the region about the mouth are free from eruption in scarlet fever and always involved in measles. There is moreover in scarlet fever the more sudden onset, decided sore throat, often initial vomiting, and the absence of catarrhal symptoms. The desquamation in the two affections is entirely different in character.

Grippe of the respiratory type may closely resemble the initial stage of measles. There is, however, less photophobia. The development of the characteristic rash in measles, or, even before this, of the buccal eruption, will settle the diagnosis. *Typhoid fever* occasionally exhibits a rash so abundant that that of measles is strongly suggested. I have occasionally seen such cases. (See p. 395, Fig. 93.) The diseases have however, no other symptoms in common. The rash of *typhus fever* may be much like that of measles and catarrhal symptoms attend the prodromal stage. Other symptoms, however, differ entirely. Severe and rapidly fatal cases of *cerebrospinal fever* may resemble malignant measles. Both possess the sudden development of threatening cerebral symptoms, while ill-defined eruptions with a hemorrhagic tendency may be present in either. I have known of several instances where the diagnosis between these two diseases could not be determined with certainty. The eruption of *variola* may at first resemble measles with an unusually papular rash. The error fortunately is usually that of considering measles to be small-pox. The diagnosis is generally soon apparent. The initial stage of *variola* is totally different, being marked by headache, vomiting and pain in the back, without catarrhal symptoms, while the rash is more papular and shot-like than that of measles. It is possible, too, for the prodromal rash of *variola* to resemble the eruption of measles to some extent. *Vaccinia* occasionally exhibits a morbilliform erythema. It has, however, no other symptoms suggesting measles. *Varicella* can scarcely be a source of confusion. The eruption could simulate that of measles only at the onset. The roseola of *syphilis* may resemble the rash of measles closely. Other symptoms, however, are lacking.

Various *eruptions*, not symptoms of infectious fevers, may have a close resemblance to measles. Notable among these are some of the erythemata, especially the medicamentous rashes, among these being those produced by antipyrine, copaiba, cubeb and chloral. That following the administration of diphtheria-antitoxin occasionally awakens suspicion. In none of these are other rubeolous symptoms present. They may, however, lead to errors in diagnosis. This is especially true if coryza happens to coexist. Urticaria, too, may exhibit at times a very morbilliform eruption. The absence of other symptoms and the general history of the attack are usually conclusive.

Treatment. Prophylaxis. Quarantine.—Although measles is generally a mild disease, yet the inability to predict the outcome in any given case necessitates the employment of all prophylactic measures possible. Especially are young children and those in a debilitated condition to be protected from it. Unfortunately the ease and frequency with which it is communicated before its existence is suspected render these measures frequently of no avail. Other children of the family who have been in contact with an affected child during the state of invasion have probably already contracted the disease. Still, as it may chance that infection may not yet have occurred, isolation should be established immediately, and maintained until all danger of infecting others is over. The appearance of the hypolymphocytosis during incubation can be made use of effectively in epidemics to enforce prompt isolation of the patient. In the absence of entire certainty regarding the duration of infectiousness quarantine should continue for at least 2 weeks from the onset, or longer if there has been a persistence of nasal or aural discharge. Other non-immune children who have been exposed should be kept from intercourse with susceptible playmates for 14 days in order to permit the usual period of incubation to elapse. It must be recognized, however, that the improbability of the disease being transmitted by a third person renders immune children of no danger to others, even if they have been associating with the patients. If the germ, as already stated, does not live longer than 2 weeks from the onset of the attack, disinfection of the room after the disease is not imperative, provided it is thoroughly cleansed and aired. There is no necessity, too, of destroying books, toys and the like. However, disinfection can do no harm, and is an additional precautionary measure. So, too, it is wise to allow the room to be unoccupied by non-immune persons for a week or more after the patient is removed from it. Other susceptible children of the family need not necessarily be sent from the house. They are not likely to contract the disease after isolation is established.

Protective treatment by inoculation has been tried by Home,¹ Thomson,² Herman³ and others with the purpose of preventing or of mitigating the attack. The success has not as yet been sufficient to be convincing.

Treatment of the Attack.—In average uncomplicated cases very little treatment of any kind is required and, in any event, is purely symptomatic. The choice and management of the sick-room, and the method of conducting quarantine and nursing are described in the introductory remarks under Infectious Diseases (p. 306). There is particular need of an abundance of fresh air without exposure in this disease, since the tendency to the development of bronchopneumonia is certainly checked in this way. The air should be somewhat moist, and the temperature of the room may well be rather higher than for certain other diseases, owing to the existence of catarrhal symptoms. The eyes should be carefully guarded from undue exposure to light, but without making the room nearly dark as is often done. The sensations of the patient are the best guide in this respect. These can be determined in young subjects by careful observation. Shielding the eyes by a screen is often a great relief. The patient should be confined to bed while fever lasts and should be lightly covered. The head must not be kept too low, as this often tends to increase the amount of cough. The diet should be light, milk

¹ *Loc. cit.*

² Glasgow Med. Journ., 1890, XXXIII, 420.

³ Arch. of Pediat., 1915, XXXII, 503.

being one of the most serviceable foods. Such cereals as oatmeal are to be avoided on account of the possibility of exciting the diarrhea to which the disease predisposes.

The patient should receive ablution daily, exposure being carefully avoided. Daily examinations of the mouth, nose and throat ought to be made in order to discover the onset of any complication here.

Further treatment of some of the individual symptoms may be considered more in detail.

Fever in ordinary cases may be combated by such simple febrifuges as potassium citrate or spirits of nitrous ether, in some cases with the addition of small amounts of tincture of aconite root. A warm tub bath is often of great benefit. If the temperature is unusually high and is unrelieved by these measures, antipyrine or phenacetin may be employed, small repeated doses being given rather than larger single ones. These drugs are, however, seldom required. The mere occurrence of a temperature of 104°F. (40°C.) at the height of the disease, unattended by any unfavorable symptoms, does not demand energetic treatment. The continued application of an ice cap to the head is often serviceable. Occasionally in obstinate and threatening hyperpyrexia the cool pack or cool tubbing is of value, unless general cyanosis or coldness of the extremities shows the existence of cardiac weakness. It must be remembered that children often bear the application of cold water or ice badly.

Nervous symptoms, such as headache, unusual restlessness, grinding of the teeth, stupor, impending convulsions and the like, are benefited by some bromide salt, and, still more, by warm tub-baths, phenacetin, or antipyrine given as described.

Constipation may be relieved by enemata. Purgatives should, as a rule, be avoided on account of their tendency to act too freely in this disease.

Diarrhea generally requires no medication in ordinary cases in healthy subjects. If at all severe, or if the child is debilitated, it must be promptly checked with bismuth, sulphuric acid, or tannic acid preparations with or without opium.

Vomiting seldom needs other treatment than temporary rest of the stomach with careful selection of the diet.

Irritation of the eyes demands the protection from light referred to. In addition there should be frequent douching with a tepid solution of boric acid. Rubbing the edges of the lids with petrolatum prevents their adhesion during sleep. Cold wet cloths applied to the eyes and changed every few minutes are serviceable when inflammation is severe. Protection and treatment of the eyes must be continued during convalescence until the irritation has disappeared.

Inflammation of the throat and nose may well be treated by spraying with an alkaline antiseptic, such as liquor sodii boratis comp. (Dobell's solution) diluted, or with a mentholated oily spray (menthol 2 grains, liquid petrolatum 1 oz.) (0.13; 30). The systematic employment of this procedure probably lessens the chances of the development of diphtheria, as well as of pneumonia through the extension of germs to the lungs. The distressing *cough* requires small repeated doses of deodorized tincture of opium, or, in older children, of heroin or codeine, enough being given to produce some result. When *bronchitis* is severe, the chest being full of small mucus râles and dyspnea present, the application of mustard poultices, or even of dry cups may be helpful. Frequently repeated warm tub baths are often beneficial, and belladonna is

also useful in this connection. The patient should be propped up well in bed, as this renders respiration easier. *Pneumonia* should be guarded against by the careful avoidance of all exposure, especially after fever has disappeared, the supply of an abundance of warm, fresh, moist air, the rigorous confinement of the child to bed, the control of bronchitis, and the use of a nasal and pharyngeal spray. It is also advisable not to keep subjects with measles and bronchopneumonia in the same room with other patients suffering from measles alone, as the pulmonary condition tends to spread to the latter.

Cardiac or general debility calls for the use of alcoholic or other stimulants. The hot mustard tub bath is of especial service in the cases where retrocession of the rash shows the weakness of the heart. The development of *otitis* may be hindered by the use of the antiseptic spraying of the nose and throat referred to, combined with the constant wearing of a flannel cap over the ears.

Itching of the skin is relieved by the frequent application of a powder consisting of camphor 1 dr. (3.9); zincum oxidum $\frac{1}{2}$ oz. (15.5); amylum $\frac{1}{2}$ oz. (15.5) or of a 2 per cent. carbolyzed petrolatum. In the case of young children a 1 per cent. thymolated petrolatum is safer.

More detailed treatment of the various complications referred to, as well as of others not mentioned in this connection, will be found described in discussing these diseases. It may be remarked, however, that the frequency with which diphtheria associates itself with measles in institutions, and with which germs resembling the Klebs-Loeffler bacillus are found on the mucous membrane of cases of measles, even though without suspicious symptoms, justifies the preventative inoculation with antidiphtheritic serum which has been recommended.

During *convalescence* the diet may be increased, but confinement to bed should continue, except in the mildest cases, for at least 10 days from the beginning of the stage of invasion; certainly for several days after fever has disappeared; and to the room for $2\frac{1}{2}$ to 3 weeks. The eyes often require careful protection for some time, and the bronchitis which frequently persists may need appropriate treatment. Before leaving the sick-room a disinfectant bath may well be given (p. 243). Owing to the sensitiveness of the mucous membranes the first outing should be in good weather and the patient carefully protected. Tonics are often required at this period, among the most useful being iron, cod liver oil and strychnine. If health is not rapidly regained, a complete change of air to a warm, dry region is advisable, owing to the tendency to the development of tuberculosis as a sequel.

CHAPTER IV

RUBELLA

(German Measles)

The disease has very numerous synonyms. The term *Rubella* was first employed by Veale¹ and appears to be that most suited to it. "Rötheln," a title formerly often applied to it is a foreign word, and there is no need for its employment in English-speaking countries.

For many years the disease had been confounded with measles and with scarlet fever, but its independence is now fully recognized. It is probably the mildest of the acute infectious fevers. The first clear description of

¹ Edin. Med. Jour., 1866, 404.

its symptoms as we now know them was given by Heim¹ in Germany and by Maton² in England. Much earlier than this accounts were published of an affection claimed to be distinct from measles, yet clearly much more severe than, and different materially in many respects from, rubella as seen at the present day.

Etiology. Predisposing Causes.—Among these *age* is important. The disease is most common between the ages of 5 and 15 years, yet adults are frequently attacked, and F. Seitz³ reports an instance in a woman of 73 years. Among 664 cases collected by J. Seitz⁴ 45 (7 per cent.) were adults. Whether adults possess a relatively lesser degree of susceptibility is uncertain, although it has seemed to me probable. Satisfactory data are lacking on this point. Infants, especially under the age of 6 months, are much less frequently attacked.

Sex, race, and climate are not etiological factors. Epidemic influence is particularly marked, the disease being unusual, even in large cities, except in certain years when extensive epidemics occur. Season is not without influence, most epidemics appearing in winter and spring. Concerning the *individual susceptibility* there is a difference of opinion. Some regard the disease as only mildly contagious, many persons seeming immune. My own experience is that it is decidedly contagious, although less so than measles, and that a large proportion of children exposed will contract it. Older subjects, as stated, probably possess a greater degree of immunity.

Exciting Cause.—This is undoubtedly a germ, but its nature is entirely unknown. *Transmission* is probably by way of the secretion from the eyes and the oral and respiratory mucous membranes, and possibly in the scales of the epidermis, although the latter is doubtful. Indirect infection through a third person, clothing, and the like, is perhaps possible, but certainly very unusual. Transmission by the air probably occurs to a very limited extent. Hess⁵ attempted to transmit the disease to apes without success.

The *period of greatest infectiousness* is unknown. The disease is, without doubt, infectious during the stages of eruption and of invasion, and it seems likely during the latter part of incubation, but probably ceases to be so with the disappearance of the rash. Exact data are lacking regarding the infectiousness during desquamation. It is doubtful whether it exists.

Pathological Anatomy.—There are no lesions characteristic of the disease. The eruption appears to depend upon a capillary hyperemia of the upper layers of the corium with slight inflammatory exudate (Thomas).⁶ More exact studies have not been made.

Symptoms. ORDINARY COURSE. Period of Incubation.—One of the features of the disease is the variability of this period. The average range would appear to be from 1 to 3 weeks, although I have seen it as short as 5 days.⁷ There are no symptoms characteristic of this stage.

Period of Invasion.—In the great majority of cases prodromal symptoms are either absent or so insignificant that they are overlooked. They usually consist of slight cough, sneezing, mild congestion of the

¹ Hufelands Journal, 1812, III, 61.

² Med. Transact. Col. of Phys., Lond., 1815, V, 149.

³ Bayrisch. ärztl. Intelligenzbl., 1873, XX, 756.

⁴ Correspondbl. f. Schweiz., Aerzte., 1890, XX.

⁵ Arch. of Int. Med., 1914, XI, 913.

⁶ Ziemssen's Handb. spec. Path.-anat. u. Therap., B. II, H. II, 128.

⁷ N. Y. Med. Rec., 1887, July 2 and 9.

mucous membrane of the nose and eyes, drowsiness, malaise, enlargement of the superficial cervical glands and sometimes slight fever. These last generally not more than 12 to 24 hours or less.

Period of Eruption. *Rash.*—Commonly without warning the child on waking in the morning is found to be covered by the characteristic rash. This appears, as a rule, first upon the face and spreads very rapidly over the body, covering it within a few hours or a day, and involving sometimes the soles and the palms. It consists of irregularly shaped, pale-rose spots, slightly elevated both to sight and to touch, and varying in size from an ordinary pin-head up to a split-pea. The spots are more or less closely placed, but not arranged in groups as in measles. They are for the most part discrete, but often show decided confluence on the face and on regions pressed upon and kept warm in bed, such as the nates and the flexor surfaces of the thighs. On the trunk the rash is usually paler than elsewhere and of a slightly brownish-red color. Very commonly the eruption passes rapidly like a wave over the body, having almost faded from the face, neck, and trunk by the time the full development is reached on the extremities 12 to 24 hours later; the acme on any one part lasting only from a few hours to half a day. This has been considered a characteristic of the disease. Nearly, or fully as often, however, in my experience, the eruption reaches its fullest development everywhere on the 2d day, after which it fades with great rapidity. The total duration of the rash upon the body equals 2 to 4 days, although it often lasts a much shorter time.

Other Symptoms of the Eruptive Stage.—Appearing with the eruption, or continuing in an accentuated form from the stage of invasion, when this is discoverable, are the trifling symptoms of the eruptive stage. They consist of moderate redness of the eyes, occasionally sneezing, sore throat, and slight hoarseness and cough. Decided coryza is absent. Often there are no catarrhal symptoms whatever, except the moderate affection of the throat, which is one of the most characteristic and constant symptoms. Sometimes this is not complained of, but on inspection the mucous membrane of the pharynx exhibits a diffuse redness, and the tonsils are generally swollen. In a considerable number of cases an eruption of yellowish-red or brownish-red spots (enanthem), of pin-head size, is visible over the soft palate, the uvula, and the mucous lining of the cheeks. This appears simultaneously with the rash upon the skin and lasts about half a day. Widowitz¹ claims to have found the buccal eruption of measles in 10 out of 135 cases of rubella (7.41 per cent.), and Müller² has also seen it repeatedly. The experience of observers in general does not corroborate this statement. I have seen 1 instance in which I believed it to be present. Occasionally, the cough and sore throat continue longer than the cutaneous eruption. Elevation of temperature is generally slight or absent. It reaches its maximum on the 1st or 2d day of the eruptive stage, and either falls suddenly while the rash is still at its height or diminishes more slowly as this fades. The fever seldom exceeds 101° to 102°F. (38.3° to 38.9°C.). Not infrequently a subfebrile temperature persists for some days after the rash has disappeared. The tongue is clean or slightly coated; never "strawberry" as in scarlet fever. Enlargement of the superficial cervical and posterior auricular glands is nearly always present and very characteristic, and often continues after the rash has disappeared. It is not, however, as

¹ Wiener klin. Wochenschr., 1889, XII, 919.

² Münch. med. Wochenschr., 1904, LI, 98.

diagnostic as once supposed, as it may occur in measles as well. Itching and edema are cutaneous manifestations occasionally seen, and roughness of the skin resembling cutis anserina is common. The bowel movements and urine are unaffected. Vomiting is unusual. The condition of the blood demands further study, since the statements are somewhat at variance. Hildebrant and Thomas¹ found an increase in the mononuclear cells and a diminution of the neutrophiles most marked from the 4th to the 6th day. Spieler² observed an increase in the neutrophiles during incubation but a diminution of these and an increase of the lymphocytes in the stage of eruption, the condition in this respect resembling that seen in measles. Hess³ noted definite increase of the number of lymphocytes before the eruptive stage.

The duration and character of the general symptoms are, as a rule, proportionate to the persistence and intensity of the eruption, but to this there are numerous exceptions.

Period of Desquamation.—Spots of a faint brownish or yellowish color are often left after the eruption has faded. These persist 2 or 3 days. A faint, branny desquamation resembling that of measles is very common but by no means always present. It appears shortly after the disappearance of the eruption and continues 1 to 3 days.

VARIATIONS FROM THE ORDINARY FORM.—The symptoms just described are what might be called typical. One of the chief characteristics of rubella, however, is its tendency to variations, many of which occur so frequently that they cannot be called unusual. On the other hand the severe epidemics described by the older writers and by a few more modern ones are to be considered as entirely anomalous.

The variable length of *incubation* is a normal and characteristic element of the disease. Epistaxis and sore throat have been reported as occasionally seen in this period (Squire).⁴ The *invasion* is sometimes unusually prolonged. I have known it to last 48 hours, and periods as long as 6 or 7 days have been reported. All such prolongations are decidedly exceptional. The prodromal symptoms may rarely be unusually severe. Among those reported are decided coryza, vomiting, convulsions, delirium, bleeding from the eyes, ears or nose, edema of the face, dizziness, fainting, severe headache, rigors, croup, and various cutaneous eruptions.

The *eruption* is extremely prone to vary, not only in different epidemics, but in different cases in the same epidemic, and even in different parts of the body in one person. In fact, this multiform character is one of the greatest characteristics of the disease. Two types are especially noticeable, which are best designated as *Rubella morbilliforme* and *Rubella scarlatiniforme*. Both must be considered as entirely normal forms.

In *Rubella morbilliforme* the spots are fully the size of a split-pea and deeper colored than usual, exhibiting the tint of measles and being often characteristically grouped as in that disease. Such a rash cannot be distinguished from that of measles.

In *Rubella scarlatiniforme*, on the other hand, the eruption, at first macular and discrete, becomes by the 2d day widely confluent and is not perceptibly elevated. Careful examination will usually reveal a

¹ Zeit. f. klin. Med., 1906, LIX, 444.

² Wien. med. Woch., 1915, LXV, 919.

³ Loc. cit., 913.

⁴ Quain's Dict. of Med., Amer. Ed., 1885, 1382.

few macules in the general redness, especially on the brows, wrists and fingers. Nevertheless, in many cases a diagnosis from scarlatina, based upon the eruption, is entirely impossible, especially if the case was not seen at the onset. Filatow¹ believes the scarlatiniform type to be a distinct disease and applies to it the older German title "Rubeola scarlatinosa." I have, however, in institution-epidemics of rubella, observed some children with an eruption exactly simulating measles, and others

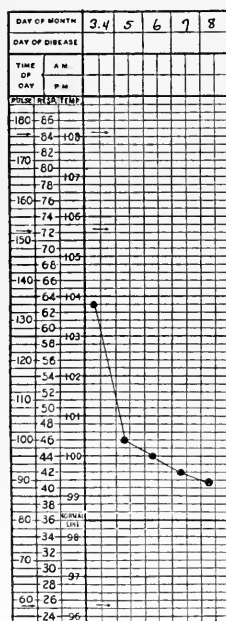


FIG. 74.

FIG. 74.—RUBEOLA MORBILLIFORME.

Katie G., aged 5 years. Mar. 4, lachrymation, injection of eyes, drowsiness, twitching of limbs, red throat, numerous discrete pea-sized macules on face; Mar. 5, rash fading from face, abundant on body, deep red, pea-sized, grouped as in measles and indistinguishable from that eruption, child brighter; Mar. 6, rash fading rapidly; Mar. 7, branny desquamation beginning.

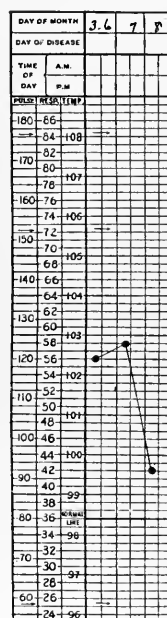


FIG. 75.

FIG. 75.—RUBEOLA SCARLATINIFORME.

Bertha L., aged 8 years. Mar. 6, eyes injected, throat red, abundant nearly uniform eruption over body suggesting scarlatina, but with macules visible in many places; Mar. 7, rash indistinguishable from scarlet fever except at wrists and ankles, throat red; Mar. 8, fading rapidly, macular character visible on legs, throat still quite red; Mar. 9, rash practically gone, branny desquamation beginning.

with the most typical rubella scarlatiniforme. All gradations may be found between the two extremes. Sometimes the rash is scarlatiniform in some regions of the body and morbilliform in others. In general, the more elevated the rash, the greater the tendency to the morbilliform type; the less elevated, the more disposition to confluence. The two illustrative cases (Figs. 74 and 75), both rather severe, illustrate these two forms of the disease. The children were inmates of the same institution and were ill at the same time, in company with many typical cases of the malady.

¹ Arch. f. Kinderheilk., 1885-6, VII, 241.

Other peculiarities of the rash are occasionally seen. Vesicles, a purpuric eruption, a marbled appearance, and a sensation as of shot beneath the surface have been reported. I have seen the rash first appear as annular red spots. The sequence of its appearance may vary also, the trunk or the arms being first involved in some cases. Sometimes the eruption is limited to a certain region, especially the face and neck, lasting but a few hours only. It is probable also that the disease may rarely occur without eruption. At times the rash nearly disappears, to reappear after a brief interval, and in other cases there seems to be a true retrocession of the rash, with general symptoms more severe than usual.

Considerable variation may occur in the *symptoms of the eruptive stage* other than the rash. The catarrhal manifestations are in rare instances as marked as in measles. This is noticed oftenest in the rubeoloid type. Decided hoarseness is sometimes present and cough may occasionally be harassing. The tonsils may be sufficiently swollen to make deglutition difficult or impossible, and sometimes the follicles are engorged. Exceptionally the temperature may reach 103°F. (39.4°C.) or over, or vomiting may be severe. Delirium and convulsions have been observed.

The *desquamation* presents nothing varying from the type, except that in some cases it is usually abundant and prolonged.

Complications and Sequels.—These occur only rarely. Those oftenest seen are in connection with the respiratory tract. Severe *bronchitis* is sometimes witnessed, and *pneumonia* may occur as a complication or sequel. I have seen it follow in 2 instances. *Croup* and *pleurisy* have been reported. *Stomatitis* is an occasional complication. *Intestinal catarrh* is rare, although in a very anomalous severe epidemic reported by Cuomo¹ it was witnessed frequently. A secondary *sore throat* occurring rarely as a sequel has been recorded by several observers, and *albuminuria* and *nephritis* have been described as frequent in some entirely anomalous epidemics (Edwards),² but ordinarily are most unusual. *Arthritis* was seen by Wichman³ in 18 out of 75 cases, and fatal purpura hæmorrhagica by Stratford.⁴

Other *infectious diseases* may occasionally be associated with rubella as complications or sequels. Among those recorded are typhoid fever, erysipelas, varicella, diphtheria and mumps. Among other complications and sequels reported, generally in anomalous cases, are thyroid enlargement, conjunctivitis, keratitis, otitis, endocarditis, icterus, urticaria, temporary paralysis, and abscesses in different parts of the body.

Relapse and Recurrence.—*Relapse* is very unusual. Isolated cases are recorded in medical literature. It occurred 3 times in 150 cases which I have previously reported.⁵ It is oftenest seen from 1 to 3 weeks after the beginning of the first attack.

Recurrence is rare, one attack fully protecting from another. In 363 cases of rubella, Widowitz⁶ saw recurrence in but 1 instance.

Prognosis.—This is almost invariably good; certainly so unless the disease has assumed an entirely anomalous form. Certain epidem-

¹ Giorn. internaz. d. scien. med., 1884, VI, 529.

² Amer. Journ. Med. Sci., 1884, LXXVIII, 448.

³ Hospitalstidende, 1898, VI, 35.

⁴ Lancet, 1911, II, 156.

⁵ Loc. cit.

⁶ Wien. klin. Wochenschr., 1909, XXII, 1596.

ics have been reported, especially in earlier years, where the mortality reached from 5 to 9 per cent. All such occurrences are, however, very unusual. Death has generally depended upon complications, but occasionally upon the depressing effect of the disease itself. I have never seen a fatal case, although I have occasionally seen individuals decidedly ill with the disease.

Diagnosis.—The most important diagnostic signs are the variable duration of incubation, the short or absent prodromes, the slight degree of catarrhal symptoms and of fever, the presence of sore throat and enlarged superficial cervical glands, and the characteristic discrete eruption. The combination of the last with insignificant attendant symptoms is very suggestive of the disease. The importance of a correct diagnosis is great, since an error may be sufficient to spread measles or scarlet fever through a house or a school. Yet owing to the variability of the symptoms this is seldom easy and, in sporadic cases, often impossible. There is no disease of which it is more true that the diagnosis must be based upon the study of the entire complex of symptoms.

Mild cases of *measles* resemble rubella very closely. The longer duration and greater severity of the prodromal symptoms in measles and the buccal eruption will be valuable diagnostic aids. Schick¹ considers a positive tuberculin test evidence of rubella as against measles. A negative reaction is without value. It is, however, the distinguishing of mild *scarlatina* from the scarlatiniform type of rubella which is especially difficult. A longer period of incubation, the absence of vomiting, and the presence of slight catarrhal symptoms with but moderate sore throat suggest rubella; but the most important diagnostic symptoms are the presence of macules somewhere in the scarlatiniform rash, and the absence of any approach to the strawberry tongue. Later the differences in the desquamation may make the diagnosis clear. When these differential symptoms fail to render a decision reasonably certain, the only safe plan, with regard to other members of the household, is to make the provisional diagnosis of scarlet fever. When an epidemic of rubella is prevailing the problem is a much simpler one.

Treatment. Prophylaxis.—The decided contagiousness of rubella and the fact that it is communicated to others even before it is recognized, render prophylaxis exceedingly difficult. In view of this, and of the usual harmlessness and short duration of the affection, it is questionable whether isolation is worth while, if only the diagnosis has been made with certainty. The inconvenience attending separation of the sick children seems scarcely warranted by the insignificant nature of the disease. If it is determined to institute quarantine, the same course should be followed as in measles and probably for an equal length of time.

Treatment of the Attack.—This is purely symptomatic and is, indeed, seldom needed. The patient should be confined to bed while fever lasts and given a light diet. Of course should the individual case or the prevailing epidemic be unusually severe, more energetic measures are required, both prophylactic and after the attack develops.

¹ *Ergebn. d. inn. Med. u. Kinderh.*, 1910, V, 302.

CHAPTER V

THE FOURTH DISEASE AND INFECTIOUS ERYTHEMA

THE FOURTH DISEASE

(Filatow-Dukes Disease)

In 1900 Dukes¹ published the report of three series of cases which, he believed, showed the existence of a fourth eruptive fever in addition to measles, scarlet fever and rubella. He maintained that the affection generally believed to be the scarlatiniform type of rubella was in reality a distinct disorder. This was practically the view advanced by Filatow² in 1885, and which had earlier been suggested by Thomas.³ The only reason for maintaining the independence of the affection was that it apparently did not protect from scarlatina. The arguments advanced by Dukes did not seem to me⁴ at all convincing, there being no proofs given that the disease was not rubella in two epidemics and mild scarlet fever in the third. This has been practically the position maintained by Williams,⁵ Caiger,⁶ Pleasants,⁷ Poynton,⁸ and most of those who have given the matter close attention. Many physicians have accepted the new disease, but as yet no cases have been reported which prove convincingly that the affection protects from neither measles nor scarlatina, and this will be necessary before it can properly be given recognition. The question is consequently still unsettled.

ERYTHEMA INFECTIOSUM

This disease, although previously described by other observers, was first claimed to be an independent infectious disorder by Escherich⁹ in 1896. Its position as a distinct affection still remains to be established, yet it certainly appears in no way to protect from scarlatina, measles or rubella. An excellent review of the subject is given by Escherich¹⁰ and by Shaw,¹¹ the latter accompanied by colored plates. Epidemics have also been described by Michalowicz,¹² Heisler¹³ and Tobler.¹⁴ The disease has not yet been observed in English-speaking countries as far as I am aware.

It occurs in epidemics especially in spring and summer (Tobler, in winter) and generally attacks children between 4 and 12 years of age. Adults also contract it, but no case has as yet been observed in infants under 1 year. It is only feebly infectious. The nature of the germ is unknown.

¹ Lancet, 1900, XXXIX, 89.

² Arch. f. Kinderh., 1885-6, VII, 241.

³ Ziemssen's Cyclop. Pract. Med. Amer. Ed., 1875, II.

⁴ Phila. Med. Journ., 1902, April 12.

⁵ Brit. Med. Journ., 1901, II, 1797.

⁶ Brit. Med. Journ., 1901, II, 590.

⁷ Phila. Med. Journ., 1902, May 24, 938.

⁸ Brit. Med. Journ., 1901, II, 594.

⁹ Transac. 11th Internat. Med. Cong., 1896.

¹⁰ Monatsschr. f. Kinderheilk., 1904, III, 285.

¹¹ Amer. Journ. Med. Sci., 1905, CXXIX, 16.

¹² Przegl. pedj., 1909, IV-V. Ref. Jahrb. f. Kinderh., 1910, LXXI, 235.

¹³ Münch., med. Woch., 1914, LXI, 1684.

¹⁴ Berl. klin. Woch., 1914, LI, 514.

Symptoms.—The period of incubation, as far as yet determined, varies from 6 to 14 days. Initial symptoms are generally absent or consist only of malaise, discomfort and sore throat. The rash appears first on the face in the form of a rose-red efflorescence distinctly raised, occupying especially the cheeks, symmetrical in distribution, disappearing momentarily on pressure, and suggesting erysipelas. The skin is swollen and hot, and the edges of the affected area are generally slightly raised and sharply defined in an irregular line. There is no itching. The forehead and chin exhibit only discrete small patches of bluish-red color, and the temples and the oral circle are uninvolved. On the 2d day the eruption appears on the body, especially the gluteal region where it is always well-developed, and on the outer surface of the arms and legs, but only to a limited extent on the trunk. In these regions it is less intensely colored than on the cheeks and resembles rather the spots on the forehead. It is morbilliform in character, but more macular than papular, with some confluence on the extensor surfaces of the arms, while on the flexor surfaces of the arms and legs it is always less intense. The hands and feet are the last parts to be involved. In many regions on the extremities the rash has a map-like appearance, especially while fading. On the trunk there are scattered discrete spots, pea-size or larger, sometimes crescentically grouped; or there may be no eruption whatever.

The rash fades from the face in 4 or 5 days, and disappears entirely from the body in 6 to 10 days from its first appearance. No pigmentation or desquamation follows. The mucous membranes exhibit no eruption.

There are practically no symptoms attending the eruption, the lymphatic glands are not swollen, there are no catarrhal manifestations, the urine is normal, fever is rarely present, and the child generally feels perfectly well.

In the matter of **diagnosis**, the disease is most likely to be confounded with rubella, although it is in reality quite unlike it. In the latter disease the rash spreads uniformly over the body and is well developed in the oral circle and on the forehead and exhibits either discrete small macules or a widespread confluence over the cutaneous surface. The lymphatic glands are enlarged. None of this is true of infectious erythema. Measles may resemble it closely, yet superficially, in the appearance of the eruption on the extremities. The involvement of the mucous membrane in this disease, the presence of buccal or palatal eruption, the distribution of the rash, and the fever and catarrhal symptoms serve to distinguish it. Scarlet fever has but slight resemblance. The general symptoms, the appearance of the throat and the distribution of the rash are characteristic in this disease. The medicamentous and other erythemata have little in common with infectious erythema, and the history, course and distribution of their eruptions aid in distinguishing them.

The **prognosis** is entirely favorable, there are no complications and sequels, and **treatment** is not required.

CHAPTER VI

VARIOLA

(Small-pox)

Small-pox is now seen but seldom in children where the practice of vaccination is prevalent, yet some description of it is requisite on account of the danger of confounding its modified form (varioid) with variella.

It has existed since earliest times in Asia and since at least the 7th century in Europe. The best early description of it was by Rhazes in the first part of the 10th century.¹ It appeared in America in the 16th century.

Etiology. Predisposing Causes.—Race, climate and locality exert no influence, the disease being spread over all parts of the earth. Sex and age, too, do not affect its occurrence. The infant may be born suffering from the disease, or having already passed through an attack, or it may develop it soon after birth as easily as at any later period of life. The individual susceptibility is very great, and the majority of unprotected persons exposed contract the disorder. Yet an immunity; temporary or permanent, is found occasionally to exist. The affection is particularly prone to appear in large epidemics, which, at least in temperate climates, reach their maximum during the cold months of the year.

Exciting Cause.—The disease is one of the most contagious known, yet the nature of the germ has never been positively determined. It seems possible that it is a protozoan (the cytoryctes variolæ), described by Guarnieri,² Councilman, Magrath, and Brinkerhoff,³ and others; but it is claimed by Proescher⁴ and others that these bodies are only proteid end-products.

Transmission.—The transmission of the germ by inoculation was long made use of in "variolation." It is transmitted also by direct contact, and, as generally believed, to an unusual degree through the surrounding air, from a distance of several hundred yards. This is, however, denied by many. It is readily carried by clothing, insects and the like, or by a third, healthy person. The infection certainly resides in the pustules and the crusts. It does not appear to be contained in the mucous secretions unless contaminated by pustular discharge. The blood, however, has been proven to be infectious and it is also evident that the disease can in some way readily be communicated before the eruption appears. Danger of infection by the patient ceases by the time the crusts have fallen and the skin has become smooth. The *tenacity of life* of the germ is very great and may continue even for some years. The poison is generally received by way of the respiratory tract and sometimes by the digestive.

Pathological Anatomy.—The earliest step in the formation of the pock is a circumscribed hyperemia followed by a necrosis of the epidermal cells, beginning in the Malpighian layer. The cells now fuse into a reticular framework, as seen later in the vesicle, and transudation of

¹ De variolis et morbillis, Lond., 1756.

² Centralbl. f. Bakt., 1894, XVI, 299.

³ Journ. Med. Research, 1903, IX, 372; 1904, XI, 12.

⁴ New York Med. Journ., 1913, XCIV, 741.

serum takes place into the lesion, with a proliferation of cells in the periphery. The combination of these changes forms the *papule*. Continued transudation of fluid produces the *vesicle*. Most investigators believe that umbilication results from the holding down of the central portion of the surface of the vesicle by the reticular bands beneath it, while the cellular proliferation of the periphery raises this portion higher. The increasing tension later tears away the reticulum and the umbilication disappears. Effusion of blood may take place into the lesion. Leucocytes in constantly increasing number enter the vesicle and a *pustule* results. They may penetrate too, the tissues forming its base, until the whole thickness of the skin is involved. If this occurs, *scarring* results.



FIG. 76.—DISCRETE SMALL-POX.

Occurring in an unvaccinated girl. 8th day of eruption. (Welch and Schamberg, *Acute Contagious Diseases*, 1905, Pl. XV, opp. p. 179.)

Analogous changes take place in the mucous membrane of the mouth, throat, eyes, nose, larynx, trachea, bronchi, esophagus, rectum, vagina, ureter, and urethra. Sometimes a pseudo-membrane covers the ulcers produced. Petechiæ may be present on the mucous membrane as well as on the skin.

Hemorrhage may be discovered in the serous membranes, the various organs, the muscles, and the medullary cavities of the bones. This is especially true of the hemorrhagic type of the disease. Hypostatic pulmonary congestion and bronchopneumonia are common post-mortem lesions. In cases which have passed into the suppurative stage, parenchymatous degeneration of the liver, heart and kidneys is found, and the spleen and lymphatic glands are enlarged.

Symptoms.—The symptoms vary to such a degree with the type of the disease, that it is more convenient first to describe the

ordinary form (*Variola vera*) and then the modifications which may arise.

(A) **VARIOLA VERA. DISCRETE SMALL-POX** (Fig. 76).—In this, the usual form of small-pox, the lesions are discrete throughout, or confluent on the face and hands only.

Period of Incubation.—This is usually 12 to 14 days, although it may vary from 5 up to 20 or more. As a rule no symptoms are present, but occasionally the last days of this period are marked by malaise, dullness, headache, and loss of appetite.

Period of Invasion.—The onset is generally sudden, with repeated vomiting, prostration, headache, pain in the back, limbs, and epigastrium, loss of appetite, coated tongue, thirst, high fever and rapid pulse. Con-

vulsions often usher in the attack in early life. A chill may occur in older children. Diarrhea may replace the constipation present in adults. The temperature rises very rapidly to 103° or 104°F. (39.4° or 40°C.) or more. There is restlessness, either sleeplessness or drowsiness, and often delirium.

On the 2d day the pain in the back and head and the rapid pulse persist, and occasionally the convulsions, vomiting and abdominal pains also; and the temperature continues unaltered or even rises. Not infrequently, but less often in children, a *prodromal rash* is observed, which is scarlatiniform or morbilliform in type. The scarlatiniform rash is generally limited to the inner surface of the thighs, the lower part of the abdomen, the axillæ, and the sides of the chest; but sometimes covers the entire surface and in color suggests erysipelas. The morbilliform eruption is irregularly distributed, either in limited areas or more widely spread. The prodromal rash may disappear before the characteristic eruption of the disease develops, or may last for a time after its appearance. Petechiæ are sometimes seen in cases destined to be hemorrhagic. The duration of the period of invasion is from 2 to 3 days. The general symptoms of the 2d day continue unabated until the eruption begins to develop.

Period of Eruption. *The Rash.*—On the 3d or sometimes the 4th day from the onset of the initial symptoms the characteristic eruption begins to show itself. The *individual lesion* consists at first of a red macule which gradually becomes more prominent and is transformed into a hard, elevated papule by the 2d day (the 4th of the disease). It now gives the often-described sensation as of a shot beneath the skin. On the 3d day of the eruption (the 5th of the disease), a minute vesicle appears in the centre and grows slowly to the 5th or 6th day, when it is as large as a good-sized split pea, very firm, of a mother-of-pearl color, circular, slightly flattened on top, often with a central umbilication, and with a narrow red areola. Pricking with a needle allows a small quantity of serum to exude, but the vesicle does not collapse; that is to say, it is multilocular.

By the 6th day of the eruption (the 8th of the disease) suppuration begins and proceeds rapidly, and by the 8th or 9th day is at its height, the vesicle having become a yellow, entirely opaque pustule, of globular form without umbilication, and surrounded by a very distinct areola with much swollen skin about it. The pustule may rupture in a day or more or may remain unruptured, while a secondary umbilication develops.

The regions first attacked are generally the face and wrists whence the rash extends to the head, hands, and arms, and in 24 hours to the trunk and then to the lower extremities, occupying about 3 days before it reaches its fullest extent. By this time the entire surface, but especially the face and head, is well covered, the rash having become vesicular in the region first attacked, while still macular or papular elsewhere. The parts of the body constantly exposed to the air show the greatest number of lesions, the trunk being nearly always least involved. In adults the distal portions of the limbs are more affected than the proximal, but this is less well-marked in children. The lesions are separated from each other by skin that is normal except for the swelling, which is especially marked on the face, hands and feet. In some cases the pocks appear everywhere simultaneously; in others first on some other regions than the usual ones. They may number some hundreds in an average case.

Those upon the palms and soles consist at first of macules surrounded by an indurated area. They become flattened, deep-seated vesicles, but do not pass through a papular stage.

Simultaneously with the cutaneous eruption, or a little earlier, lesions may be found on the mucous membrane of the mouth, pharynx, nose, and sometimes the larynx, vagina, rectum and eyes. These run a course similar to those upon the skin, except that the vesicles soon rupture and leave ulcerated surfaces.

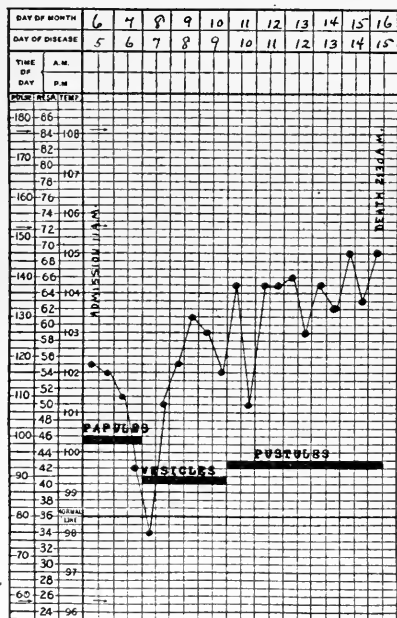


FIG 77.—DISCRETE SMALL-POX WITH TYPICAL COURSE.

Emily E., aged 5 years. Never vaccinated. Mar. 2, nausea, headache, aching in legs and back, fever; Mar. 5, amelioration in symptoms, papular rash developing on face; Mar. 7, decided fall of temperature, vesicles appearing; Mar. 10, rise of temperature with pustular stage. From a patient in the Philadelphia Hospital for Contagious Diseases. Courtesy of Dr. B. Franklin Royer.

Symptoms Attending the Eruption.—With the appearance of the rash upon the skin, or shortly after or before it, there is usually a sudden or gradual fall of temperature to normal or nearly so, and the constitutional symptoms improve greatly (Figs. 77 and 80). When suppuration begins on the 6th day of the rash, the temperature gradually rises again with morning remissions. This is called the "secondary" or "suppurative" fever. Then follows increase of temperature until about the 11th or 12th day of the eruption when it falls by lysis in favorable cases, the elevation reached being generally less than in the stage of invasion. With the onset of the secondary fever the pulse and respiration again become rapid, the heart often weak, and prostration decided. There is severe pain, swelling, tension, and itching of the face, neck and extremities. The eyes may be closed and nasal respiration obstructed; the face so swollen that the patient is unrecognizable. Restlessness, sleeplessness, and delirium are common. The throat is sore and swallowing difficult, thirst excessive, the tongue parched, the breath foul, the teeth and gums covered with sordes,

diarrhea may occur, and conjunctivitis and cough are common. The urine often contains albumin and casts. The discharging pus from ruptured lesions produces a very offensive odor. The pain which the swelling of the skin occasions at this period is often intense and every movement or even the pressure of the bed or the bed-clothes may be the cause of great suffering.

The blood in small-pox exhibits a diminution of hemoglobin early in the disease. The red blood-corpuscles tend to form irregular clumps instead of rouleaux. Magrath, Brinkerhoff & Bancroft¹ confirm the

¹ Journ. Med. Research, 1904, XI, 247.

observations of previous investigators, that a varying degree of leucocytosis, especially of the mononuclear cells, is present during the eruptive stage.

Period of Desquamation.—Drying begins about the 11th or 12th day of the eruption, generally first in the lesions which were first to appear. By the 14th or 15th day, in average cases, this process is well under way, and the crusts begin to fall. Many, however, are very adherent and do not separate until during the 4th week or later, unless removed forcibly. Following the separation a branny desquamation occurs. On the hands

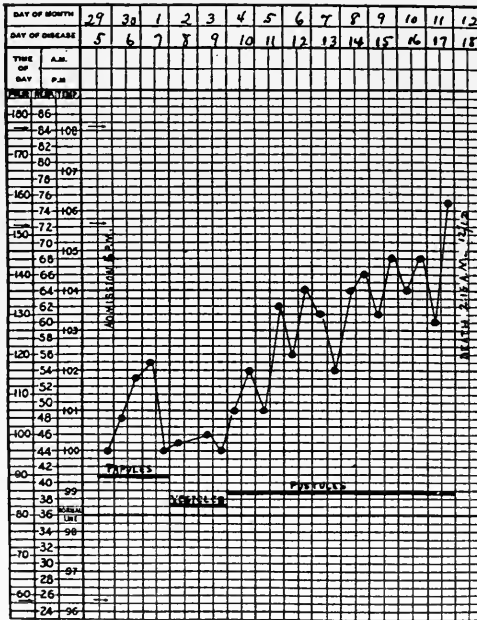


FIG. 78.—CONFLUENT SMALL-POX.

Adult. Never vaccinated. June 25, very intense headache and backache, with dizziness, vomiting, chills, and high fever; June 28, been very ill and in great pain, widespread papular eruption appeared this date and spread rapidly; July 1, vesiculation began, lasted 3 days, became confluent on face, legs, wrists, and forearms; July 4, pustulation began, great pain and high fever which lasted until death. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

and feet the entire skin may be shed in the form of moulds. After desquamation is over there remains a reddish pigmentation, later becoming brownish. The normal color is regained only after weeks or months.

With the beginning of desiccation the swelling and pain in the skin lessen, the areole about the pustules grow smaller, the temperature begins to fall and the symptoms in general improve. The itching of the skin is now frequently intense. The hair often falls out temporarily. Feebleness of body and of mind may persist for several weeks and disappear only slowly. Any scarring left by the disease does not assume the characteristic white appearance for 3 or 4 months.

The principal well-defined modifications of the type of the disease are (B) *Confluent small-pox*, (C) *Hemorrhagic small-pox*, (D) *Mild small-pox*, (E) *Varioloid*, or *small-pox modified by vaccination*.

(E) **CONFLUENT SMALL-POX.**—This form is characterized by the tendency of the lesions to fuse, generally with an increase in severity of all the constitutional symptoms. The prodromes are the ordinary ones, but always severe. The eruption appears at the usual time or often somewhat earlier, and spreads with great rapidity. The lesions are closely crowded, and the swelling and redness of the skin come on early. Sometimes the face assumes a general redness at the beginning of the eruptive period instead of exhibiting the ordinary macules. In the suppurative stage many of the pustules fuse into irregularly shaped suppurating areas, especially on the face and extremities, but on the trunk they continue discrete. The entire face may be covered by one enormous pustule. The involvement of the throat is usually extensive.

The subsidence of symptoms and the fall of temperature seen as the eruption is developing are less than in discrete small-pox. Then, with the onset of suppuration or before it, all the general symptoms are manifested in great force. Vomiting and retching may be continuous, the cervical lymphatic glands are much enlarged and the temperature is high with little remission (Fig. 78). The symptoms in general are those of intense toxemia. The crusts which form later are slow in being shed.

(C) **HEMORRHAGIC SMALL-POX.**—This is the malignant form of the disease. The incubation is often very short. The hemorrhagic tendency may develop either in the prodromal or in the eruptive stage. In the former, called **purpura variolosa** or “black small-pox,” the symptoms are violent and a purpuric rash appears, at first petechial and later ecchymotic. Much of the surface of the body may exhibit an almost uniform purplish color. Hemorrhage from various mucous membranes may occur, oftenest from the renal. Generally the true eruption of small-pox does not develop; or, if the patient live until the time of the eruptive period, is represented by a few papules only. There may be delirium or stupor, but often the mind remains clear throughout. This is the most malignant form of the disease.

In the second variety of hemorrhagic small-pox, called **variola pustulosa hemorrhagica**, the earlier stages of the disease are severe, but there is nothing characteristic until the vesicles or pustules begin to form. The hemorrhagic tendency then develops either slowly or rapidly. Bleeding takes place into the lesions and, in the still severer cases, into the surrounding tissues and from the mucous membranes. The mind usually remains clear.

(D) **MILD SMALL-POX.**—Individual cases, or even epidemics, occur in which, although there has been no protection by vaccination, the course of the disease is extremely mild. The lesions are few in number or entirely absent (**Variola sine eruptione**), and the constitutional symptoms mild or insignificant. Occasionally the prodromes are of the usual severity, but the development of the eruption is abortive, and the whole course of the disease greatly curtailed, the condition being entirely similar to varioloid.

(E) **VARIOLOID; MODIFIED SMALL-POX.**—This is the form observed in persons partially protected by vaccination. It is characterized by its mildness and the comparatively small number and rudimentary nature of the lesions. The disease, however, does not differ in nature from ordinary small-pox and is as capable of producing the unmodified form in unprotected persons. The stage of invasion is more variable than in discrete small-pox, ranging often from 1 or 2 days up to 4 or 5 days. The prodromal symptoms may be the ordinary ones of small-

pox or may be mild and even overlooked. A prodromal rash is frequent, especially of the rubeoloid form. The distribution of the lesions is very variable. Sometimes only about half a dozen may be found, or even exceptionally none at all. They are often confined to the face and hands. In other cases they may be quite numerous or may even be confluent or partially so. As a rule they develop first on the face and spread rapidly. Their first appearance is that of red macules, suggesting the roseola of typhoid fever. These quickly change into papules and then some of them into vesicles, while others never pass the papular stage. Many of the vesicles do not suppurate but rapidly dry into crusts; others become purulent, but are superficial, soon dry, and rarely leave scars. The

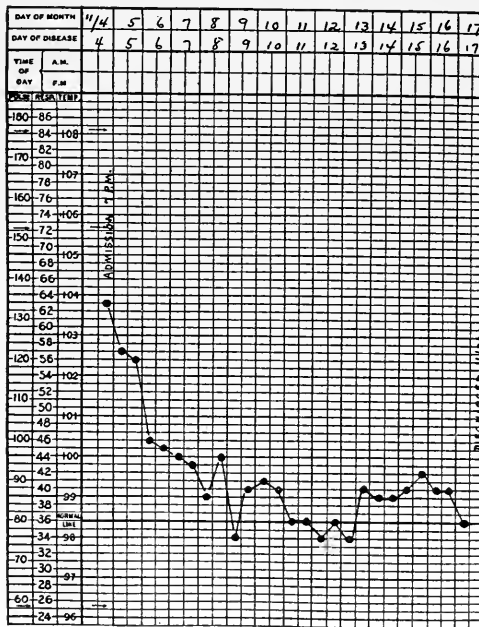


FIG. 79.—VARIOLOID.

Adult. Vaccinated in youth. Nov. 1, headache and backache, high fever, nausea, dizziness; Nov. 3 symptoms continued until this date, when generalized papular eruption developed; Nov. 4, vesicles developed, but nearly all dried in this stage, a few pustules without secondary fever. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

entire crop of lesions also develops more rapidly than in unmodified small-pox and is present within 12 hours from the first evidence of eruption. There are no large areolæ and no severe swelling of the skin. With the first appearance of the eruption the initial fever falls rapidly by crisis and is seldom again elevated, the suppurative fever being absent and all symptoms disappearing. The whole process is much shorter than in variola vera and the desiccation begins earlier and is soon completed (Fig. 79).

Complications and Sequels.—Affections of the respiratory apparatus are among the most frequent complications of variola. Laryngitis is common, and in severe attacks involvement of the cartilages may occur, or edema of the glottis cause death. Bronchitis is invariable in

severe attacks of small-pox and bronchopneumonia is almost always present in fatal cases. Lobar pneumonia is uncommon, but pleurisy, especially of the purulent form, is frequent. A pseudodiphtheritic inflammation of the throat is not uncommon. Cardiac complications are rare, myocarditis, occurring oftener than other forms. Otitis is observed occasionally and conjunctivitis, keratitis, and iritis not infrequently, sometimes followed by more or less permanent impairment of sight. Albuminuria is common, but nephritis is rare. Multiple cutaneous abscesses are of frequent occurrence as sequels (Fig. 80). Bed sores or gangrene may develop, and suppurative adenitis is not infrequent.

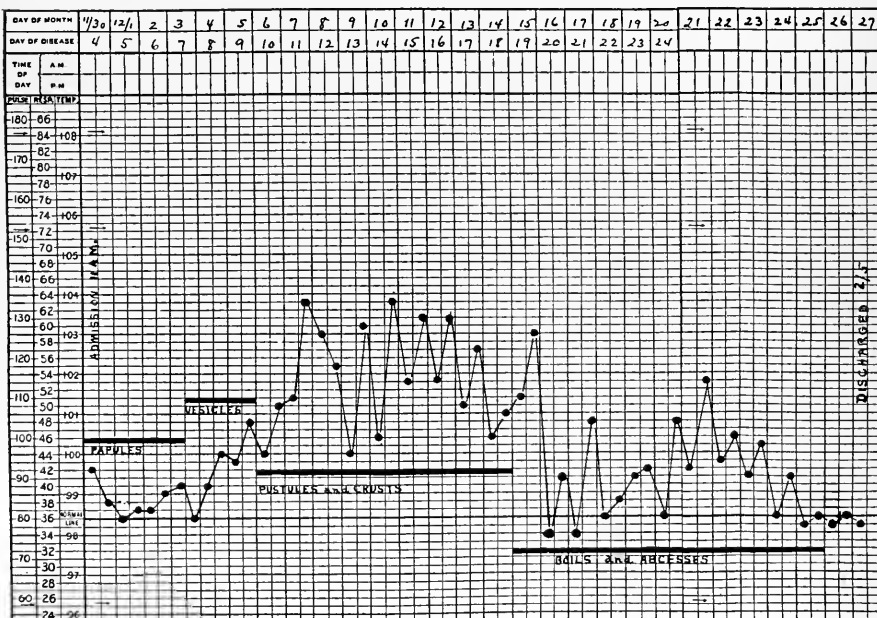


FIG. 80.—DISCRETE SMALL-POX FOLLOWED BY BOILS AND ABSCESSES, PROLONGING THE TEMPERATURE.

Adult. Usual prodromes began Nov. 27 and patient was severely ill for 3 days; papular eruption on Nov. 30, with rapid subsidence of all symptoms; boils and abscesses appeared on Dec. 16. From a patient in the Philadelphia Hospital for Contagious Diseases. *Courtesy of Dr. B. Franklin Royer.*

Among nervous disorders neuritis is an occasional sequel and myelitis, encephalitis and meningitis may occur as complications.

Small-pox may coexist with, follow or precede other acute infectious diseases but this appears to be very exceptional. Erysipelas may develop as a sequel during convalescence.

Relapse and Recurrence.—*Relapse*; i.e. a second attack developing before the poison is out of the system is rarely observed. *Recurrence* of small-pox is sometimes seen, but is very uncommon. As a rule one attack confers a life-long immunity, and a second occurrence is usually mild. Vaccination gives an immunity which may be permanent or only temporary. (See Vaccination, p. 380.)

Prognosis.—The mortality of unmodified small-pox is high, varying from 25 to 45 per cent. or more. The character of the epidemic is one of the important factors. In some outbreaks the virulence is but slight,

and few cases die. Various causes influence it. In infancy and early childhood the number of deaths is very large. Statistics of the Hospital for Contagious Diseases of Philadelphia (Welch and Schamberg)¹ gave a mortality of 61.66 per cent. in subjects under 1 year, as compared with a total mortality of 31.79 per cent. A mild initial stage generally presages a mild attack. Severe initial symptoms, however, do not necessarily indicate that the attack as a whole will be severe. Death may occur in any stage of the disease. In many cases the initial symptoms are so grave that children die before the eruptive stage develops. Danger again arises during the suppurative period, and it is at this time, about the 11th or 12th day of the disease, that the majority of deaths take place. A widespread, intense, scarlatiniform prodromal eruption is an unfavorable symptom, often indicating the onset of hemorrhagic small-pox. The morbilliform prodromal rash is rather a good omen. The presence of petechiæ during invasion is generally, but not necessarily, an unfavorable indication. A fall of temperature occurring promptly with the development of the rash promises a mild attack, while unusually high fever during the period of eruption is unfavorable and may signify that a fatal ending is immediately impending. The sooner the suppurative fever begins to fall the better the prognosis and the less danger there is of threatening complications developing. Severe nervous symptoms during the eruptive period are an unfavorable indication, as is an abundant eruption on the mucous membrane of the mouth and throat. The danger of the disease is usually in direct proportion to the number of pocks and the amount of suppuration. Confluent small-pox gives a very high mortality, especially under 5 years of age. The great majority of hemorrhagic cases die. The prognosis in modified small-pox is nearly always good, depending upon the degree of protection which vaccination has afforded.

Diagnosis.—In well-developed small-pox the diagnosis can scarcely fail to be made. Early in the disease, however, or later in very mild cases, and especially in the modified form, it may be very difficult. The characteristic diagnostic symptoms are the severe pain in the head and back in the initial stage, the sudden drop of temperature about the beginning of the eruptive period, and the well-marked papular stage through which the eruption passes. Early in its development the rash has often been mistaken for *typhoid fever*. At this period, too, it may strongly resemble *measles*, and errors in diagnosis have frequently been made. The absence of the catarrhal symptoms of measles, however, and of the Koplik spots aid in excluding this. The morbilliform prodromal eruption of small-pox may likewise suggest measles. This eruption, however, is not at all elevated as is the rash of measles. The scarlatiniform prodromal rash may lead to the suspicion of *scarlet fever*. The absence of initial sore throat, and the localization of the eruption elsewhere than first on the face and chest, serve to exclude the latter disease. Further, the prodromal rashes are not common in children. I have seen a *generalized vaccinal eruption* occasion confusion. The development of the rash at the same time with the vaccine vesicle and the absence of all the characteristic initial symptoms of small-pox render the diagnosis generally easy, yet not always so, as the vaccination-pocks of vaccinia may occur in combination with that of small-pox (Fig. 81). The *pustular syphiloderm* also may be mistaken for small-pox. The slower onset and course, the ap-

¹ Acute Contagious Diseases, 1905, 275.

pearance of the eruption in successive crops, the history, and the presence of other evidences of syphilis aid in recognizing this affection. *Varicella* occurring in a severe form is the disease causing the greatest diagnostic difficulty. This is especially true in the exceptional cases where the stage of invasion of varicella has been unusually long or well-marked. So, too, mild varioloid may closely simulate ordinary varicella. In varicella, however there is a rapid development and drying of the vesicles, and in small-pox, even in the modified form, the invariable presence of a papular stage and a slower desiccation. In varicella, again, is the occurrence, side by side, of lesions in all stages of development, and in variola the existence of but a single crop in any one locality. The rash of vari-

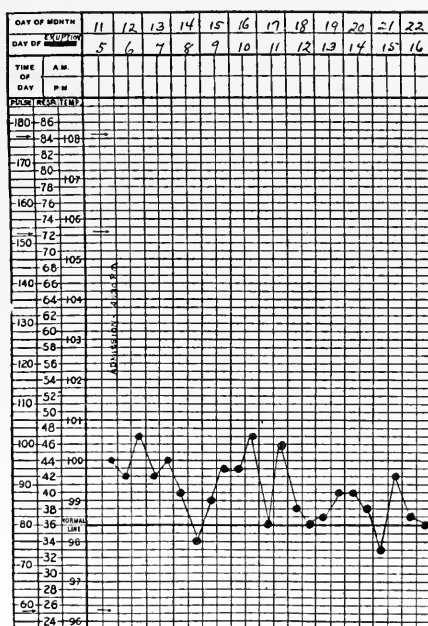


FIG. 81.—MODIFIED SMALL-POX AND VACCINATION OCCURRING TOGETHER.

Philip C., aged 7 months. Exposed to small-pox from Oct. 24 to Nov. 1. Vaccinated Nov. 1; began to take actively by Nov. 4; eruption of small-pox appeared Nov. 7, but dried in vesicular stage. Fever probably due in part to active vaccination. From a patient in the Philadelphia Hospital for Contagious Diseases. Courtesy of Dr. B. Franklin Royer.

cella is most abundant on the trunk, especially in the back; that of variola on the face and the distal parts of the extremities. These differences make the diagnosis generally clear. Yet in spite of them mistakes are frequent and easily made.

Treatment. Prophylaxis. Quarantine.—Owing to the intense infectiousness of the disease, any suspected case should be isolated absolutely at once. The extreme diffusibility of the poison renders quarantine in private houses very difficult, and treatment in special hospitals is to be highly recommended. When this is impossible the patient should be confined to his room in the method recommended for infectious diseases in general (p. 307), and all non-immune persons should leave the house. These must necessarily be kept under surveillance and

away from other persons until a time equalling that of the period of incubation is past. Everyone exposed should be promptly vaccinated, since by its more rapid development vaccination may modify or even prevent the action of the small-pox infection (Fig. 81). The isolation of the patient must continue until every vestige of the disease has disappeared. This takes longest on the thick epidermis of the palms and soles. The time is necessarily very variable but averages 4 to 6 weeks from the onset of the disease. The patient, the room, and every article which has been exposed should then receive the most thorough disinfection. The patient should be given a bath of 1 : 10,000 corrosive sublimate solution and the room be thoroughly fumigated with formalin. Attention to detail cannot be too minute.

Treatment of the Attack.—This is necessarily purely symptomatic. The patient should be kept scrupulously clean. The bed-clothing should be light and changed often, the room well ventilated and moderately cool, the food light but nourishing and administered frequently. Water should be given freely. The aching of the initial stage may require opiates, and the fever may need to be controlled by suitable hydrotherapy. The free use of cardiac stimulants is indicated if there is any evidence of weakness. Such sedatives as the bromides and chloral may be required, if great nervous excitement exists.

Many methods have been recommended for the treatment of the cutaneous eruption. None possesses undoubted power to check the tendency to pitting, although the cutting off of the sun's chemical rays by the exposure to red light only has had much said in its favor. Welch and Schamberg¹ believe some good has been accomplished by the employment of tincture of iodine. It may be diluted if necessary. Much can be done to alleviate the tension and itching of the skin by the application of cold, wet dressings changed frequently, or of glycerin and water, or oily substances. The employment of a mask to keep these in place is often serviceable. Except for young children the oily dressings may contain carbolic acid. Ichthyol (5 to 10 per cent.) has been highly recommended for local use. It is important to keep the crusts always moist with aqueous or oily substances, and frequent warm bathing is useful in favoring their softening and removal as well as earlier for the relief of the cutaneous irritation.

The local conditions of the mouth, throat and nose require astringent and cleansing gargles, washes and sprays. The holding of ice in the mouth or the employment of orthoform or cocaine locally often gives great relief. The eyes must be cleansed several times a day with boric acid or other mild antiseptic solutions. During the suppurative stage the importance of abundant nourishment is very great and free stimulation is often required. The same methods, with general tonic treatment, are frequently needed during convalescence. Modified small-pox generally requires little treatment of any sort, especially after the initial stage is past.

¹ *Loc. cit.*, 311.

CHAPTER VII

VACCINIA. VACCINATION

Etiology and Pathology.—Vaccinia, or cow-pox, is an infectious eruptive disease oftenest seen upon the udders of cows, although the horse is occasionally subject to it. Its identity with small-pox has been disputed, but it seems most probable that it is this disease modified by its occurrence in cattle. The inoculation of the human subject with the vaccine virus is called *vaccination*. Although it had long been a popular belief that the contracting of cow-pox from animals prevented small-pox in man, and although the inoculation with the vaccine virus had been practised previously, yet the careful testing of the actual protective power and the urging of the procedure upon the medical profession is to be credited to Jenner.¹

The microorganism of vaccinia is not definitely known. Various bacteria have been discovered in the lymph, and certain bodies, apparently protozoa, and entitled "cytocytes," have been found in the serum of the vesicle by Guarnieri² and others. Paschen³ claims, however, that these are only end-products, and that the germ is much more minute and still undetermined. This is sustained by Prowazek and Miyazi.⁴ Whatever its nature, it must be the same as that of variola.

Vaccination was originally performed with matter taken from a cow-pox sore occurring upon a human being. Shortly afterward the virus obtained directly from the cow was employed. Later both bovine and humanized lymph were extensively used, but of recent years the former has entirely supplanted the latter. This has removed the possible danger of transmitting disease, especially syphilis, by the use of humanized lymph. The lymph may be dried upon ivory points or quills, but is preferably, and now usually, mixed with glycerin and preserved in glass tubes; the so-called "glycerinated lymph." Glycerin destroys all pyogenic bacteria if thorough ripening has been allowed. Noguchi⁵ has devised a method of inoculation of the testicles of rabbits and young bulls with a virus free from bacteria, and of making cultures from the inoculated organ. In this way virus absolutely free from foreign germs can be produced.

Age for Vaccination.—Regarding the age when vaccination is best performed there is some difference of opinion. That which I prefer is about 3 or 4 months. By this time the vigorous infant will probably have overcome any earlier digestive difficulties. After this age the constitutional effect of vaccination is liable to be greater. Should small-pox be prevalent vaccination should be done in the 1st month. Other circumstances, too, influence the decision, and an occasion should be selected when the infant is in good general condition and free from eczema, and when the weather is not very hot.

Method.—It has been largely recommended to vaccinate in two or three different spots about an inch apart, on the ground that the greater

¹ An Inquiry into the Causes and Effects of the Variolæ vaccinae, etc., London, 1798.

² Centralbl. f. Bakt., 1894, XVI, 299.

³ Münch. med. Wochenschr., 1906, LIII, 2391.

⁴ Centralbl. f. Bakt. und Parasitenk., 1914-15, Orig., LXXV, 144.

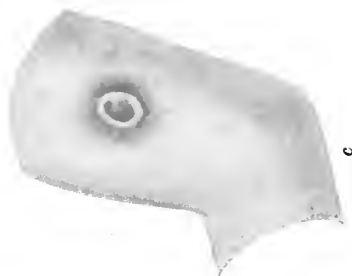
⁵ Arch. of Pediat., 1915, XXXII, 698.



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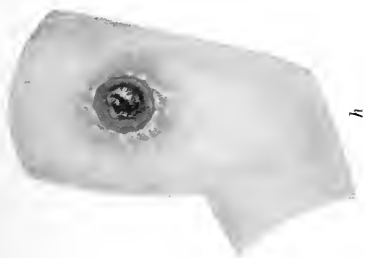
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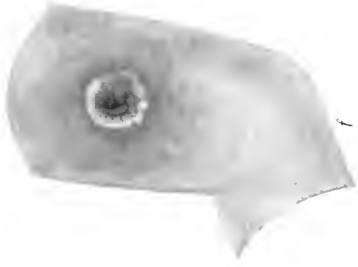
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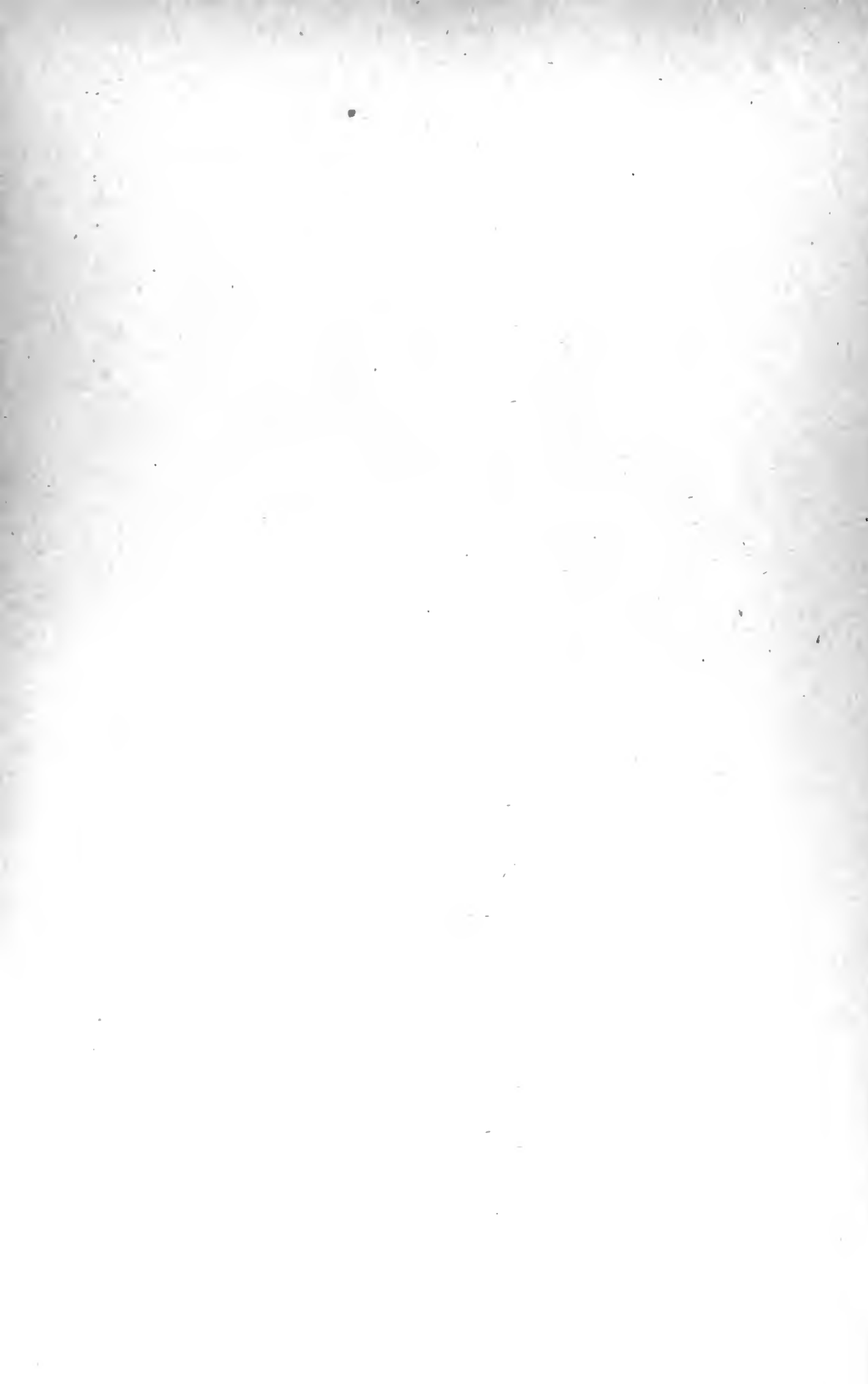


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FIG. 82.—COURSE OF NORMAL VACCINATION.
(*a*) 5th day; (*b*) 6th day; (*c*) 7th day; (*d*) 8th day; (*e*) 9th day; (*f*) 10th day; (*g*) 11th day; (*h*) 15th day; (*i*) 25th day.



the number of lesions the less will be the subsequent danger of contracting small-pox. In America scarifying in a single spot is more commonly employed. (See Protective Power of Vaccination, p. 380.) The *locality* usually chosen is the outer surface of the upper arm at about the insertion of the deltoid muscle, or, in the case of female infants, if the presence of scarring is objected to, the outer surface of the leg shortly below the knee. The skin should be vigorously cleansed with soap and water and then with alcohol, and the hands of the operator disinfected. A needle previously sterilized by being boiled or by holding the point in an alcohol flame, should be used to make on the skin four or five scratches parallel and close together, and then an equal number crossing them. They should be only deep enough to scarify the epidermis and expose the blood-vessels, but not to draw blood, lest the entrance of the virus be prevented by it. The whole surface of scarification should not exceed one-quarter of an inch. By compressing the small rubber bulb which is commonly sold with the glass tube the lymph flows out upon the wound, and may then be gently but thoroughly worked into the scratches. In place of the needle a scarifier may be employed, as used for the cutaneous tuberculin test. Very complete and satisfactory outfits are now furnished in which the glass tube terminates in a sharp point with which the scarification may be done. The point is then broken off and the upper end of the tube as well, the rubber bulb fitted and the virus expelled. The wound should be allowed to dry for 15 or 20 minutes and then be covered with salicylated absorbent cotton bound upon it with a gauze bandage, and made firm with a few strips of rubber adhesive plaster. The cotton should be left constantly in position throughout the whole course of the development of the pock, unless the vesicle ruptures and moistens it too greatly, or an unusual degree of suppuration takes place. In this case the application of fresh cotton is needed. During the vaccination-period the infant should be sponged but not placed in the tub, in order that the cotton shall not become wet. Vaccination shields are not to be recommended.

It has been advised by several writers to vaccinate by means of intradermal injections, since in this way the scarring is absent or lessened. The method has not appeared to possess sufficient real advantage and has not been widely adopted.

Course of Normal Vaccination (Fig. 82).—The development and the histological structure of the vaccine lesion is practically identical with that of small-pox. (See p. 365.) Except for the scratch marks the skin shows no alteration until the 3d or sometimes the 4th or 5th day. This closes what may be called the *period of incubation*. A small, faintly red macule now appears and marks the beginning of the *period of eruption*. Infiltration quickly changes this into a papule surrounded by a narrow red areola. By the 5th day of the vaccination a small vesicle appears in the papule, and by the 6th day this covers the scarified area and is filled with transparent lymph. The vessel is multilocular in structure and umbilicated, resembling that of small-pox. By the 7th or 8th day it reaches its maximum size, is of a pearly-grey color and half an inch (1.3 cm.) or less in diameter. The areola about it then commences to extend in width and the lymph to become cloudy through the production of pus cells in it. The vesicle is now tense and yellow, the umbilication disappears, the areola grows still more pronounced, and by the 9th day the underlying and surrounding tissues are red and swollen. On the 10th or 11th day desiccation begins and the surrounding swelling and redness diminish rapidly.

The pock is now flaccid and depressed, and rapidly dries into a dark crust. This crust is often fully formed by the 15th day, although it does not fall off, unless forcibly removed, until about the end of the 3d week or later, when it leaves a reddish scar which afterward becomes white, depressed, and characteristically pitted (Fig. 83).

Certain *symptoms* attend the vaccinal eruption, but less often in the early months of life than later. Some itching and tension may occur when the papule is developing and still more when the pustule is at its height. About the 5th day after vaccination there is in many cases



FIG. 83.—SCARS OF VACCINATION.

Four good scars from an infantile vaccination, showing pitting and depression beneath surrounding skin. Patient contracted in adult life a mild, modified smallpox for which he was treated at the Philadelphia Hospital for Contagious Diseases. Stains of smallpox lesions visible in the photograph. (Welch and Schamberg, *Acute Contagious Diseases*, 1905, Pl. II.)

slight fever of a remittent type, which gradually increases, reaching its maximum of 102° to 104°F. (38.9 to 40°C.) on the 8th or 9th day with the full development of the pustule and then falling quickly or gradually (Fig. 84). There is fretfulness, restlessness, loss of appetite, malaise and occasionally vomiting. The axillary or the inguinal glands may be swollen and tender when the pustule is at its height. A decided leucocytosis is present (Sobotka).¹

Irregularities in the Course of Vaccination.—Not infrequently the incubation is unduly prolonged, the lesion not making its appearance

¹ Zeitschr. f. Heilk., 1893, XIV, 349.

under a week or even more after vaccination. Less often incubation is shortened and the vesicle is visible by the 3d day. The fever is in some cases decidedly high. The local process may be severe, the lesion becoming unusually large, and not infrequently having several small secondary vesicles developing in the surrounding tissue. These generally run an abortive course.

In rare cases a **spontaneous generalized vaccinia** is witnessed (Fig. 85). In this condition secondary vesicles are found on various parts of the body. They first appear at the time of the maturation of the original pock, or shortly before or after this, and then continue to occur in successive crops, maturing rapidly, the whole process extending occasionally

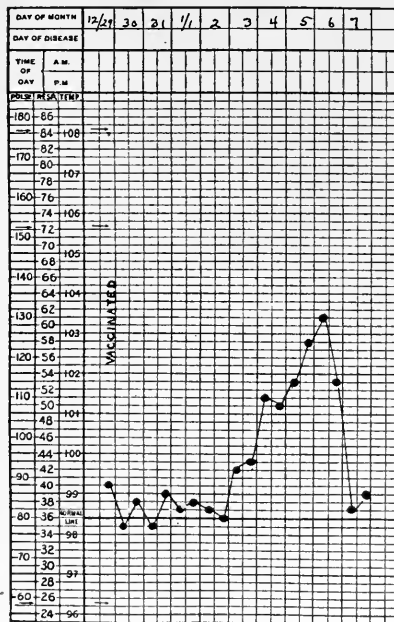


FIG. 84.—VACCINATION.

Marie B., aged 21 months. Dec. 29, vaccinated; Jan. 3, vomited; Jan. 4, lesion well developed; Jan. 5, arm very sore, axillary glands swollen; Jan. 6, fretful and restless during night, arm unchanged; Jan. 7, arm much less swollen and child bright.

over several weeks, and consequently exhibiting at the same time different stages of development. Generally they leave no scars, but to this there are exceptions. In some cases the lesions strongly suggest variola.

A **vaccinia generalized by autoinoculation** likewise may occur, the child reinoculating itself in various places by scratching or in other ways. The spots are usually very few in number. If eczema, for instance, is present the region involved may become infected from the vaccine pustule and a secondary large confluent lesion develop. The lesions have been seen in the mouth, throat, eye, tongue, nose and elsewhere. The title "generalized vaccinia" is better reserved for the spontaneous form.

Generalized vaccinia is fortunately rare, inasmuch as it may affect the entire system severely and even cause death. Huddleston¹ was able

¹ Med. News, 1901, LXXIX, 370.

to collect from medical literature but 50 reported cases of generalized vaccinia, most of them of the spontaneous form, 7 of which terminated fatally. Fatal cases have since been reported by others (d'Astros;¹ Hegler²).

Spurious Vaccination.—Particularly in subjects vaccinated not for the first time a lesion may appear which is not evidence of a genuine vaccinal infection. In some such cases a vesicle develops promptly and rapidly produces pus and crusts, the latter by the 7th or 8th day. The attendant itching is severe. In some cases merely a papule forms, or a very small vesicle upon this, no scar resulting. In others a projecting,



FIG. 85.—SPONTANEOUS GENERALIZED VACCINIA.
Primary vaccination below the knee.

dark-red, slightly granular "raspberry exerescence" may develop instead of the normal vesicle. In these conditions it is probable that little, if any, protective power results, and revaccination should be practised.

Revaccination and Insusceptibility to Vaccination.—As with all infectious diseases certain individuals are temporarily or permanently immune. Repeated vaccinations during infancy may fail to take. In all such cases further attempts should be made at intervals, as there is no certainty that the insusceptibility will be lasting.

Protective Power of Vaccination.—Nothing except the widespread employment of vaccination can account for the great diminution in the number of cases of small-pox. Not only do general statistics

¹ Marseille méd., 1912, XLIX, 149.

² Dermatolog. Wochenschr., 1914, LVIII, Ergänzungsh., 29.

throughout the civilized world prove this beyond cavil, but the instances of severe local outbreaks of small-pox in regions where vaccination had been neglected show both the importance of the procedure and that the virulence of small-pox has by no means lessened with the passing years. In countries where vaccination has been made compulsory its value is particularly marked. The accompanying illustration (Fig. 86) shows graphically the result. After the introduction of compulsory vaccination into Prussia in 1875, the average annual death-rate from small-pox fell to 1.91 per 100,000 of the population, whereas from 1816 to 1870 it had ranged from 7.32 to 62.0 per 100,000. In Austria without such regulation the contemporaneous death-rate from it was unaffected (Welch

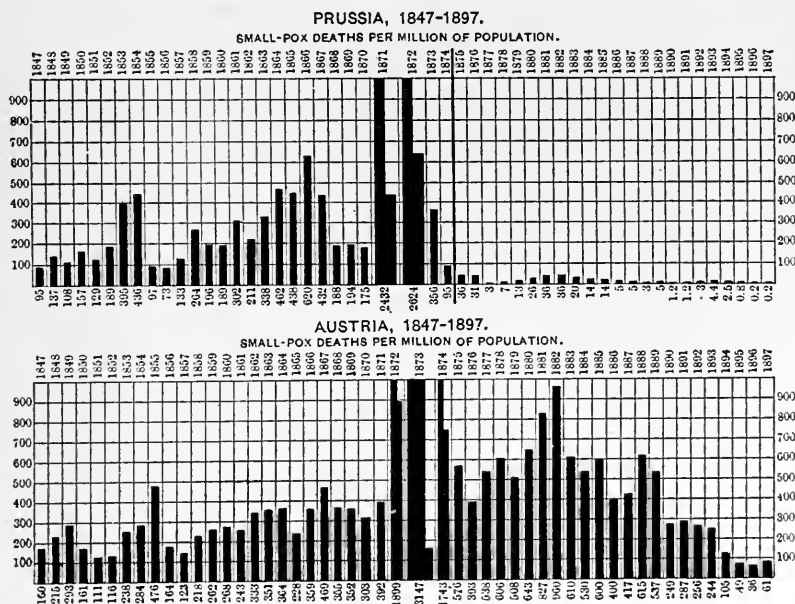


FIG. 86.—TABLES SHOWING THE DECLINE OF SMALL-POX IN GERMANY AFTER THE ENACTION OF COMPULSORY VACCINATION IN 1874.

Smallpox mortality is compared with that of Austria. (Welch and Schamberg, *Acute Contagious Diseases*, 1905, 123.)

and Schamberg).¹ In the Philippine Islands where small-pox had previously been rampant, with an average annual mortality of at least 6000 cases in the six provinces near Manila, not a single vaccinated person died after systematic vaccination was established in 1907 (Heiser and Oleson).²

The duration of the protective power varies. With some persons it is complete and permanent, while with others it gradually lessens, and modified or even typical small-pox is acquired if exposure occurs, the severity of this depending to some extent upon the time which has elapsed since vaccination was performed. The naming of an average duration of protective power is impossible. Welch and Schamberg³ estimate that absolute protection by vaccination in infancy is found in only about 25 per cent. of the cases. It is safe to maintain that revaccination should

¹ *Loc. cit.*, 123.

² U. S. Publ. Health Rep., 1911, XXVI, pt. 1, 277.

³ *Loc. cit.*, 115.

always be performed at the age from 5 to 7 years, again at puberty, and again at the beginning of adult life. Certainly, too, if small-pox becomes prevalent, all those who have been certainly exposed should be revaccinated at once, and everyone who has not had a successful vaccination within 5 years should submit to it. If the attempt at revaccination fails it should be repeated. If this also fails, we cannot, of course, be certain that immunity exists, but it is reasonable to assume it.

Although it is manifestly not to be expected that successful revaccination in a partially protected person will produce a lesion exactly similar to that of a primary vaccination, yet it should certainly possess some general resemblance to the original. The character and size of the primary scar has some bearing upon the *degree of protection* offered. The statistics of the Hospital for Contagious Diseases of Philadelphia, from 1891 to 1903 (Welch and Schamberg),¹ give a mortality from small-pox of 6.55 per cent. in patients with one good scar from vaccination in infancy, 14.39 per cent. in those with one fair scar and 24.83 per cent. in those with one poor scar. The degree of protection has been generally believed to be in proportion to the number of scars; hence the custom prevalent in Europe of vaccinating in several places. The statistics of the Philadelphia Hospital for Contagious Diseases do not entirely support this view, the quality rather than the number of the marks appearing to exert the greatest influence.

Complications and Sequels.—An unusual degree of inflammation may occur in the sore, depending upon trauma or a depreciated state of health, or oftener upon infection by pyogenic germs. Deep ulceration may follow, the entire thickness of the skin at the site of the lesion being destroyed, or a diffuse cellulitis or even gangrene developing. Such an occurrence is nearly always due to an infection of the wound at the time of vaccination or later; rarely to any trouble with the virus itself. An attack of eczema may sometimes be produced by vaccination. Urticaria is a not infrequent complication, and a rubeoloid erythema is still oftener seen, developing during the maturation of the pock. This may be localized, or may cover much of the body. Other cutaneous disorders have occasionally been reported as complications or sequels, among them impetigo, lichen, ecthyma, furunculosis, psoriasis, pemphigus and miliaria. In rare cases the pocks and the surrounding tissue may become hemorrhagic. Exceptionally a general sepsis may follow vaccination, arising from the infection of the wound. An interesting small epidemic of this is described by Brouardel.² Erysipelas was formerly a common and much dreaded complication, but with improved aseptic methods it has become very rare. Lotz³ found but 2 deaths reported from it in 1,252,554 vaccinations. Syphilis has rarely, but undoubtedly, been communicated by vaccination. The employment of bovine lymph entirely avoids the chance of this. The transmission of tuberculosis is perhaps possible with human lymph, but the danger with glycerinated lymph from calves does not exist. Tetanus has undoubtedly been given by vaccination, the germs either entering accidentally an ill-cared-for wound, or being contained in an infected lymph. Although McFarland⁴ collected 95 cases from medical literature, the occurrence of this disease as a complication is relatively very rare. Proper supervision of the produc-

¹ *Loc. cit.*, 57.

² Twentieth Century Pract. of Med., XIII, 534.

³ Ref. Brouardel, *loc. cit.*, 534.

⁴ Proceed. Phila. Co. Med. Soc., 1902, XXIII, 166.

tion of the lymph used will avoid it entirely, so far as the contamination of the lymph is concerned. There is, it is true, always a possibility of its production by the entrance of germs from the air into the vaccination-wound; but much less, with proper care, than exists in the case of any slight wound which has been left exposed to the air.

Mortality.—The mortality from vaccination is a negligible figure if proper precautions are taken. According to Kübler¹ there were 113 deaths in approximately 32,000,000 vaccinations (0.000035 per cent.) done in Germany during the 13 years from 1885 to 1897, and many of these were directly traceable to neglect.

Treatment.—The treatment of the vaccination lesion has already been described. Should fever develop a mild febrifuge is advisable. In subjects beyond the 1st year of life the vaccinated limb should be used with moderation. If an unusual degree of inflammation occurs the dressing must be removed and the wound treated on general surgical principles.

CHAPTER VIII

VARICELLA

(Chicken-pox)

Although described much earlier, varicella was first differentiated clearly from variola by Fuller in 1730² and by Heberden in 1767.³ There are probably still some who consider it only a form of variola, but the very great majority of writers of the present day believe it to be an entirely independent disease, basing this opinion on its appearance in distinct epidemics and its entire failure to protect from small-pox or to be prevented by vaccination.

Etiology. Predisposing Causes.—*Age* is the most important of these, varicella being preëminently a disease of infancy and especially of early childhood. It is observed in the first months very much oftener than either scarlet fever or measles (Pridham).⁴ It has even been described as occurring congenitally. Although the susceptibility lessens greatly after the age of 10 years yet the statements of many writers up to quite recent periods, to the effect that it is never seen in adult life, are contrary to the general experience. Thus, Wanklyn⁵ found that of 200 cases of varicella 16.7 per cent. were in persons over 18 years of age. Such frequent occurrence after childhood is, however, unusual.

Seasons, climate and sex exert no predisposing influence. The individual susceptibility in childhood is very great, most of those exposed contracting it. Not infrequently, however, some children seem to be entirely immune. That so few adults are attacked is probably in part due to the fact that most persons have already had the disease in early life. It is oftener endemic than epidemic. Thus in some years in large cities great numbers of cases are seen, in others much fewer; yet it is always present to some extent.

¹ Geschichte der Pocken und der Impfung, 1901, 364.

² Gee, Reynolds Syst. Med., Amer. Ed. 1879, I, 124.

³ Med. Transac. Col. of Phys., London, 1768, I, 427.

⁴ Brit. Med. Journ., 1913, I, 1054.

⁵ Brit. Med. Journ., 1902, II, 47.

Exciting Cause.—This is undoubtedly a germ, the nature of which is, as yet, unknown, although various microorganisms have been reported. De Korte¹ described an ameba-like body present in the vesicles, and Greeley² a sporothrix which corresponds to the cytoryctes variolæ. Further study is needed. The method of *transmission* is not definitely known. Although the disease is in the large majority of instances acquired by direct contact or by short exposure in the vicinity of an affected child, it is not infrequently conveyed mediately through a third unaffected person or through articles of clothing and the like. It is probable that, next to variola, chicken-pox is the one of the infectious fevers most readily transmitted mediately. Numerous inoculation experiments have been made with the serum of the vesicles, and Steiner³ claimed to have succeeded 8 times in 10 cases, but his results are exceptional, and the disease is seldom transmissible in this way. Buchmüller⁴ had negative results in 30 cases. The readiness with which varicella is spread in schools shows conclusively that the *period of infectiousness* commences at the very beginning of the eruptive stage, and, there is reason to believe, even before this. It is uncertain how long it continues, or whether the virus is present in the crusts, but the duration of life is probably short.

Pathological Anatomy.—The characteristic lesions consist solely of macules and papules developing later into vesicles, which occupy only the upper layer of the epithelium and are unilocular, or often multilocular as can be demonstrated by pricking them with a needle. The areola often surrounding them is formed by distended capillaries and slight cellular infiltration. The fluid contents are of clear serum which later becomes cloudy and contains a few leucocytes. In some of the vesicles, under the influence of irritation or infection, the process extends deeper, a large exudation of leucocytes takes place, and the corium is destroyed and replaced by scar tissue.

Symptoms. **ORDINARY FORM.**—The *stage of incubation* is somewhat variable, averaging about 14 days, but being sometimes less, and extending often up to 3 weeks; in Steiner's inoculation-cases always 8 days. The *stage of invasion* is usually absent, although occasionally there may be slight malaise, moderate fever, and general aching, present for 24 hours or less before the rash develops. At times a widespread *prodromal erythema* of a scarlatiniform or, more rarely, morbilliform type is seen a few hours before the appearance of the characteristic rash. This is generally most marked on the trunk and lower limbs. As a rule, however, the first symptom observed is the *fully developed eruption*. Tracing the history of the *individual lesion* from its onset, there is first found a red macule disappearing on pressure and of the size of a pin-head to that of a split-pea or larger. Within a very few hours a small vesicle filled with clear liquid develops upon the centre of the macule. In a much smaller number of lesions a small papule forms upon the macule, and a vesicle then very quickly develops on the apex of this. The vesicle rapidly enlarges and reaches its height within a day or less from its first appearance. In shape it is round or oval, and in size very variable, but averages that of a split-pea (Fig. 87). A narrow red areola often surrounds it. *Desiccation* now begins at once, the prominent rounded surface

¹ Practitioner, 1905, LXXIV, 50.

² New York Med. Rec., 1914, LXXXVI, 204.

³ Wien. med. Wochenschr., 1875, XXV, 304.

⁴ Mittheil. des Verein der Aerzte in Thiermark, 1886. Ref. Swoboda, Pfaundler und Schlossmann Handb. der Kinderh., 1906, I, b, 724.

flattening a little, and the contents becoming somewhat turbid. Slight umbilication is common at this stage. After 12 to 24 hours more the areola disappears, and a yellowish crust begins to form and is complete in from 3 to 4 days. It darkens and finally falls off after a period varying from 5 or 6 days to 2 or 3 weeks leaving the skin slightly reddened for a time.

This is the course in the typical lesion. The majority of the lesions, however, do not mature. Many macules never develop further, and are no longer visible after a few hours, while a few pass into the papular stage, continue so for a day or two and then disappear. Many vesicles



FIG. 87.—ERUPTION OF CHICKEN-POX, SHOWING THE PEARLY VESICLES.

From a case in the Children's Hospital of Philadelphia.

never grow larger than the head of an ordinary pin and dry very rapidly. On the other hand vesicles are often larger than the average, and require a longer time for full development and drying.

The rash generally makes its first appearance on the face and back, less often on the limbs. It spreads over the body from above downward, but so rapidly that the discovery of any sequence is difficult. All parts of the surface are attacked, including the palms and soles and the mucous membrane of the mouth and throat, especially the hard palate; of the nose and the genital organs; and rarely even of the larynx and trachea and the eyes. Upon mucous membranes the covering of the vesicle soon ruptures under the influence of moisture, and the lesion suggests that of aphthous stomatitis.

The number of lesions varies greatly from perhaps a dozen up to hundreds. A case of average intensity presents from 25 to 75. In mild

cases there may be scarcely a dozen. They are generally most numerous on the trunk, especially the back. The forearms, hands, legs and feet are involved to a decidedly less extent. The head is generally less affected than other regions but there are nearly always some lesions on the scalp. All the vesicles which mature are generally fully crusted in from 6 to 8 days from the first appearance of the eruption.

One of the principal characteristics of the eruption is its appearance in successive crops during 3 or 4 days in the regions already involved. As a result of this fact, and of the failure of so much of the rash to mature,

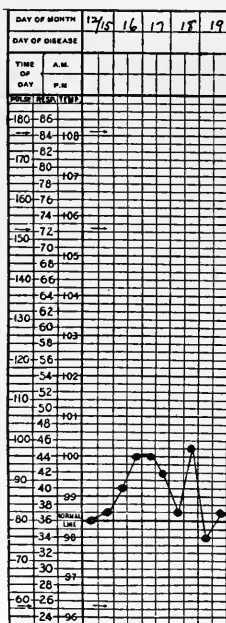


FIG. 88.

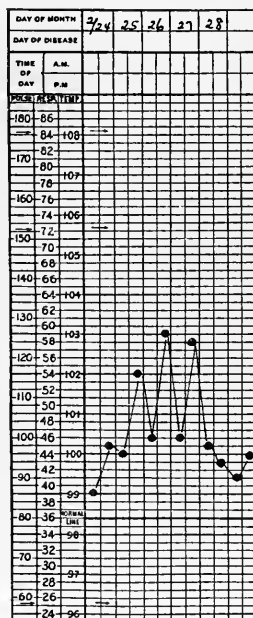


FIG. 89.

FIG. 88.—MILD VARICELLA.

Philip A., aged 2 years. Eruption developed on Dec. 17. Practically no other symptoms.

FIG. 89.—MODERATELY SEVERE VARICELLA WITH UNUSUALLY WELL-DEVELOPED ERUPTION.

Caroline R., aged 8 years. Feb. 25, a few vesicles on the body; Feb. 27, child dull; very numerous vesicles, including a large number on the tongue; Feb. 28, improving.

lesions in all stages of development from macules to crusts may be seen side by side. When a vesicle has become infected, or has been greatly irritated by the rubbing of the clothing, decided suppuration is liable to take place, and an inflamed area is produced, covered by a scab which conceals pus beneath it. The healing is thereby delayed and white scars may be left similar to those of variola.

Symptoms attending the eruption are slight or absent. Moderate fever, averaging 101° to 102°F. (38.3° to 38.9°C.) may be present during 2 or 3 days in well-developed cases, but generally falls gradually to normal in 1 or 2 days. It is higher and lasts longer if the attack is severe (Figs. 88 and 89). There may be some loss of appetite and disturbance of sleep. Considerable itching of the skin is often present. The blood shows no

characteristic alterations. The effect upon the polymorphonuclear and mononuclear cells respectively appears to be inconstant (Weill and Decos;¹ Nobecourt and Merklen;² Mensi³).

VARIATIONS FROM THE ORDINARY COURSE.—To be mentioned here are only those variations which may be considered uncommon. Among unusual *prodromes* are vomiting, delirium, convulsions, somnolence and severe headache and backache. I have known the latter in the case of an adult to suggest variola very strongly. Blood-stained stools have also been reported.

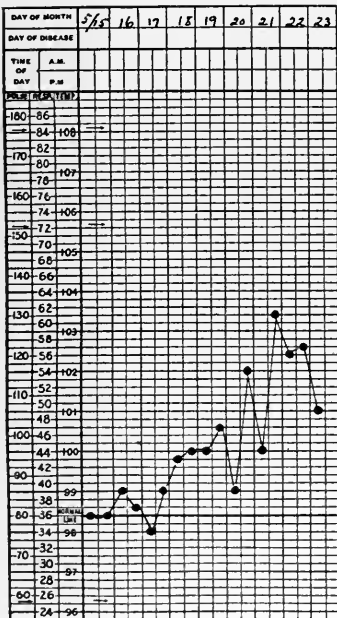


FIG. 90.

FIG. 90.—HEMORRHAGIC VARICELLA.

Elizabeth G., aged 1½ years. May 16, vesicles appearing on the back; May 19, eruption abundant and widespread; May 22, eruption very profuse and infiltrated with blood; May 23, removed from hospital on account of pertussis developing.



FIG. 91.

FIG. 91.—VARICELLA GANGRENOUSA.

Occurring in an infant of 22 months, a patient in the Children's Hospital of Philadelphia. Diphtheria followed by measles, and in a few days by varicella. Large vesicles, some sloughing, developed on whole body, especially the head and back. Death.

Distinct variations of the *cutaneous lesions* may be seen in the eruptive stage. The prodromal erythema may sometimes continue into, or first appear during, the eruptive period. A *confluent varicella* is very exceptionally observed, in which the lesions are situated on a red, swollen base common to them all, and are so closely placed that they almost touch. This is especially marked on the forehead and back, while elsewhere the discrete character of the lesions is apparent. The eyes are closed by the greatly swollen eyelids, the fever is high, the pulse

¹ Journ. de phys. et de path. gén., 1902, IV, 504.

² Journ. de physiol. et de pathol. gén., 1901, III, 439.

³ Gaz. degli ospedal. et dell. clin., 1912, XXXIII, 1625.

rapid, and the patient clearly ill. Such cases may arouse a strong suspicion of small-pox.

Hemorrhagic varicella (Fig. 90) is a rare form characterized by effusion of blood into the vesicles and sometimes from the gastroenteric mucous membrane as well. I have seen it but once or twice. The subject has been reviewed by Storrie¹ and others. The term **gangrenous varicella** has been applied to a complicating condition (dermatitis gangrenosa), which, although perhaps seen oftenest in connection with chicken-pox, may occur in other conditions as well. It develops only in debilitated, cachectic children. Certain of the vesicles, under the influence of pyogenic infection, enlarge and suppurate, and a gangrenous process develops in them destroying even the entire thickness of the skin. Septic symptoms and death are liable to follow. In one case under my observation the bacillus pyocyaneus was recovered from the lesions. In another (Fig. 91) the disease occurred in a child who suffered simultaneously from rubeola, diphtheria and varicella.² **Bullous varicella** is a very uncommon variety in which the eruption appears in the form of large bullæ which may reach an inch or more in diameter and which on rupture leave a painful raw area.

Complications and Sequels.—These are few. One of the chief in importance, although not common, is nephritis, which develops oftenest at the end of the 1st or during the 2d week of the disease. It is of an acute parenchymatous nature, the urine containing albumin and casts. Recovery generally follows promptly, but the disease may be severe enough to be fatal, or may become chronic. A varicellous laryngitis is a severe and sometimes fatal complication rarely seen. It produces symptoms of croup and may require intubation. Multiple arthritis occurring during or at the close of the eruptive period has repeatedly been described. It may be serous or purulent in character. Anemia is a not infrequent sequel. Varicella may be combined with other infectious diseases, such as measles, rubella, pertussis, scarlet fever, diphtheria, vaccinia, tuberculosis, and even variola. Erysipelas, too, may develop through infection of the lesions.

Among other conditions reported as occasionally complicating or following varicella are pneumonia, pleurisy, retention of urine the result of swelling of the prepuce, osteomyelitis, otitis, thyroiditis, symmetrical gangrene, appendicitis, multiple sclerosis, chorea, polioencephalitis, myelitis, abscesses, furunculosis, subcutaneous emphysema and adenitis. The connection of many of these with the attack of varicella seems probably entirely accidental. I have seen one case of fatal purpura hemorrhagica occur as a sequel.

Relapse and Recurrence.—*Relapse* is certainly very rare. Comby³ observed it in one instance 15 days after the drying of the vesicles. Dawes⁴ found 4 relapses in 30 cases of the disease; but such a frequency is certainly most exceptional. A few other instances are reported in medical literature which may properly be placed in this category.

Recurrence is also extremely uncommon. One attack protects from a second in nearly every instance. In 524 cases of the disease observed by Widowitz⁵ there was no instance of it.

¹ Brit. Jour. Child. Dis., 1914, XI, 62.

² Univ. Med. Mag., 1896, Aug.

³ Traité des mal. de l'enf., Grancher, etc., 2d Ed., I, 376.

⁴ Albany Med. Ann., 1902, XXIV, 532.

⁵ Wien. klin. Wochenschr., 1909, XXII, 1596.

Prognosis.—Although the disease is one of the least serious of the infectious disorders, yet fatal cases are occasionally seen. Death is usually due to complications such as nephritis, erysipelas, pneumonia and gangrenous dermatitis. Varicella of the larynx has a high mortality, as has hemorrhagic varicella. In marantic children the mere development of an ordinary varicella may not be without danger. I recall 4 such infants, sick at the same time in adjoining beds, in all of whom progress toward recovery was apparently checked by the development of chicken-pox, and a fatal issue followed. Very exceptionally in unusually severe cases death may occur during the stage of eruption; it would seem as the direct result of the toxemia of the disease.

Diagnosis.—As a rule this is easy, the eruption being so characteristic that mistakes cannot arise. It rests upon the absence or short duration of prodromal symptoms, the absence of any distinct papular stage in the great majority of the lesions, the situation of the lesions in the superficial portion of the skin, the production of a fully formed vesicle in less than a day from the first appearance of the macule, the development of the vesicles in successive crops with the resulting presence at one time and in the same locality of lesions in all stages, and the drying without, as a rule, any suppuration. Yet severe cases of varicella, especially in adults, may strongly suggest *small-pox*, and on the other hand, modified small-pox may similarly closely resemble varicella. (See Variola, p. 374.) It is to be noted that as a rule the eruption of small-pox selects especially the face and distal portion of the extremities; that of chicken-pox the trunk. To this there are exceptions. As regards *scarlet fever*, it may be difficult or impossible to determine in some cases whether one is dealing with the combination of this with varicella or with the scarlatiniform prodromal rash of the latter disease.

In rare instances the vesicular form of *impetigo contagiosa* may simulate varicella closely. The history of the attack, with the study of symptoms other than the rash, will generally eventually lead to a correct diagnosis. In other cases of varicella seen for the first time during the stage of desquamation, the resemblance to impetigo may be decided. The latter disease, however, spreads irregularly as a result of auto-inoculation by the fingers. The combination of *acne* with varicella may also obscure the diagnosis for a time. *Pemphigoid* eruptions may at first suggest the existence of varicella with unusually large vesicles.

Treatment. Prophylaxis.—Owing to the mildness of the disease it is seldom thought necessary to isolate patients suffering from it. Yet in the case of schools, in hospitals where children debilitated by other diseases are present, and even in private homes where there are delicate infants, isolation should be practised. This should be continued as long as crusts remain on the body; *i.e.* a period of 2 or 3 weeks. Prophylactic inoculation has been attempted by Kling,¹ Rabinoff² and others, the children being inoculated with the serum from a fresh varicella vesicle. The procedure is claimed to have given immunity in 75 per cent. or more of the cases.

Treatment of the Attack.—Usually little treatment is needed. The patient should be confined to bed and given a light diet as long as fever continues. The administration of a febrifuge is advisable. In severe cases the treatment is symptomatic. The urine should be examined frequently for albumin. Itching may be allayed by the application of

¹ Berl. klin. Woch., 1915, LII, 13.

² Arch. of Pediat., 1915, XXXII, 651.

a powder of camphor and zinc-oxide or of a thymolated, ichthyolated, or, in older children, a carbolated ointment; or by the employment of warm baths. Great care should be exercised to prevent scarring by irritation through scratching, and any large crusts beneath which the existence of suppuration is suspected should be removed and the lesions treated antiseptically. During convalescence the patient ought to remain in the room or in the house. Any subsequent anemia or other sequels or complications require treatment appropriate for them.

CHAPTER IX

TYPHOID FEVER

Typhoid fever, now widely disseminated and universally recognized, was first clearly distinguished from typhus fever by Gerhard,¹ using his own and Pennoek's cases, only as long ago as 1837.

Etiology. Predisposing Causes.—Climate and season exert a certain influence, the disease being somewhat more prevalent in temperate zones and in autumn and early winter. The influence of *age* is very decided. With the exception of a very few observers, it is only within about the last sixty years that typhoid fever has been recognized by physicians as occurring in childhood, and even now perhaps most authorities claim that it is uncommon in early childhood, and very rare in the first 2 years of life.

All statistics indicate that the periods in which the greatest number of cases are seen are later childhood, youth and early adult life. There is however, considerable variance of opinion regarding both the absolute and the relative frequency of typhoid fever before these periods. As compared with the number of cases observed later the disease is decidedly less frequent up to the age of 5 years, and especially in the first 2 years of life. That it cannot be called "rare" as is so often done is shown by the statistics, among others, of Barthez and Sanné² (90 cases from 2 to 4 years), Schavoir³ (68 under 5 years in a total of 406 at all periods of life) and Montmollin⁴ (15 under 2 years in 295 children under 15 years). The statistics of general hospitals are misleading, because children are usually treated at home or in special hospitals for children. Even the statistics of these special hospitals by no means represent the actual number of cases in infancy, since the majority of infants who fall ill are cared for at home. This is especially true of those in the 1st year of life. Writing in 1902 in collaboration with Dr. Maurice Ostheimer⁵ we detailed 18 cases of the disease developing in the first 2½ years of life, in our own patients or, in a few instances, in those of colleagues. We collected also from medical literature, including these cases, 139 reported as occurring in the 1st year, 187 in the 2d year, and 68 in the first half of the 3d year. While undoubtedly many of these were instances of errors of diagnosis, there have certainly existed very many more in which the disease has not been recognized, or, recognized, has not been reported.

In the spring of 1906 there were in the Infants' Ward of the Children's Hospital, Philadelphia, which contained but a small number of beds,

¹ Amer. Journ. Med. Sci., 1837, XIX, Feb. and XX, Aug.

² Mal. des enf., 3d Ed., 615, III, 373.

³ N. Y. Med. Rec., 1895, XLVIII, 803.

⁴ Observ. sur la fièvre typh. de l'enf., 1885.

⁵ Amer. Journ. Med. Sci., 1902, Nov., 868.

5 cases of typhoid fever at the same time, in subjects not over 2 years of age. Writing in 1912¹ I analyzed 75 cases occurring in the first 2½ years of life, personally attended in hospital or private practice or in the practice of colleagues in the Children's Hospital.

Typhoid fever may even be found in the fetus and the new born (*Fetal and Congenital Typhoid Fever*). In 1898 I collected from medical literature 10 such cases² which appeared to be beyond question, the germs having been recovered from the tissues or the blood. A few additional instances have since been collected (Griffith and Ostheimer,³ Morse⁴). Whether the fetus can suffer from the disease in utero, recover, and be born alive and well, is not yet established. The discovery of the agglutinative reaction in the blood of a healthy new-born infant, born of a typhoid mother and not yet suckled by her, would make prenatal recovery a possible thing, but would not be certain proof of this, since the agglutinating principle may have passed to the fetus from the placenta, without there having been any actual disease of the fetus present. (See p. 401.)

The youngest reported instance of typhoid fever acquired after birth is that by Gerhardt in an infant of 3 weeks.⁵ I have seen 1 case at 3 months.⁶

Sex exerts practically no etiological influence, although hospital statistics generally give more male cases than female. Previous good health would appear rather to favor than to prevent infection. So also favorable hygienic conditions apart from the question of the transmitting of germs exert no protective power. Statements regarding the influence of other diseases present seem to be very contradictory. In my experience they appear not to be a factor, except that very possibly dietetic errors, producing an irritated condition of the intestinal canal, may readily predispose. Typhoid fever tends to occur in *epidemics*, which, however, are more limited in locality than in the case of many other infectious fevers. In large cities it is more or less endemic. It is particularly where the disease occurs in several members of one family that children are liable to be attacked. The *individual susceptibility* seems less than that in many other infectious diseases. Whether or not the lesser frequency in infancy is due to a lesser susceptibility or to an absence of equal exposure is uncertain, but the latter seems more probable. The fact that the milk consumed is so often subjected to a high temperature and the germs consequently killed would readily account for this.

Exciting Cause.—The exciting cause of typhoid fever is the *bacillus typhosus* of Eberth, an actively motile organism of the colon-bacillus group found widely spread throughout the tissues of the body and in the blood, bile, sputum, stools and urine.

Transmission.—It is certain that in the vast majority of cases the transmission of the germs is by the feces or the urine. The feces, however, do not appear to be a favorable substance for the actual growth of the bacilli, and those found there have entered with the bile, the gall-bladder being a region where the bacilli may persist for long periods. Whether the expired air conveys the germs to any noteworthy extent is

¹ Arch. of Pediat., 1912, August.

² Phila. Med. Journ., 1898, Oct. 15.

³ Loc. cit.

⁴ Med. News, 1903, LXXXIII, 193.

⁵ Handb. der Kinderkr., II, 373.

⁶ Phila. Med. Journ., 1898, II Oct. 15.

doubtful. Water used for drinking and other purposes is the most frequent carrier, and numerous large epidemics have been traced to this source, while milk contaminated by germ-containing water, or by the infected hands of milkers, is a common carrier in early life unless it had been boiled before it was ingested. Kober¹ analyzed 195 epidemics of typhoid fever and found that the disease was transmitted in probably 148 in this way. The bacilli may also be transported from typhoid stools by flies (Vaughn)² and possibly also from the same source by the wind (Pfuhl).³

In the ordinary sense of the term, however, typhoid fever is but little contagious, and the isolation of patients is not essential. Although hospital infection has repeatedly occurred and the disease has spread in families from the sick to the well, this has not been through mere proximity, but through lack of ordinary care in disinfection. Unquestionably the soiled hands of a nurse, or garments or other articles soiled by feces and urine, can transmit the germs to the food or other objects which enter the mouths of children. The acquiring of the disease in this way occurs probably most frequently in early life. Whether typhoid fever may be contracted through germs transmitted in the milk from a mother suffering with the disease is uncertain, although the passage of the agglutinating principle is well recognized. (See pp. 391; 401.)

However transmitted the germs are probably always absorbed through the digestive tract and rapidly pass into the blood. An exception is seen in the case of fetal typhoid fever, where the transmission is by way of the placental blood. The *period of greatest infectiousness* appears to be during the 2d and 3d week of the disease, or later until healing of the intestinal ulcers has taken place, but Conradi⁴ from a study of 600 cases of all ages found the bacilli in the feces even during the stage of incubation, and believed that the infection is often transmitted during this period. The *tenacity of life* of the germ both within and without the body is often very great. It may survive under favorable circumstances for months, or even for years, and it is not destroyed by ordinary cold or by drying, but is killed by a temperature of 60°C. (140°F.), by disinfectants and by sunlight. In a case reported by Soper⁵ it seemed probable that the germ had continued present in the intestines of an individual during 5 years, and cases of the persistence for much longer periods are on record. (Dean,⁶ 29 years; Bolduan and Noble,⁷ 46 years; and others.) Such long persistence is, however, very unusual.

Pathological Anatomy.—The lesions so characteristic of the disease in fatal cases in adults are notably altered in early life. In fetal typhoid fever there is an entire absence of intestinal ulceration, perhaps the natural result of the placental mode of entrance of the germs. In the majority of congenital cases, too, intestinal lesions have not been reported, and this is occasionally true of older subjects. After these periods the lesions vary somewhat with the age of the patients. In the first 2 years of life the process is more hyperplastic than destructive, and the solitary and agminated glands are swollen, projecting, and of a pink-

¹ Amer. Journ. Med. Sci., 1901, May.

² Amer. Journ. Med. Sci., 1899, CXVIII, 10.

³ Zeitschr. f. Hyg., 1893, XIV, 1.

⁴ Deut. med. Woch., 1907, XXXIII, 1684.

⁵ Journ. Amer. Med. Assoc., 1907, XLVIII, 2019.

⁶ Brit. Med. Journ., 1908, I, 562.

⁷ Journ. Amer. Med. Assoc., 1912, LVIII, 7.

ish color. Slight and generally superficial ulceration may be present, and in some cases there is no intestinal involvement whatever. The swelling of the mesenteric glands is usually very pronounced and the spleen is always acutely enlarged and soft; and these two conditions are suggestive; but there is nothing in the post-mortem findings in infancy which is positively diagnostic of typhoid fever.

In early childhood, between the ages of 2 and 6 years, the lesions are very similar to those just described, except that the intestinal ulceration is somewhat more marked. It is still, however, generally superficial and the process is predominatingly hyperplastic. There are, however, many exceptions to this rule, and very extensive ulceration may occur even at 6 years (Fig. 92.)



FIG. 92.—ULCERATION IN TYPHOID FEVER.

Child of 6 years. Autopsy showed an unusual degree of intestinal lesions for this time of life.

In later childhood intestinal ulceration becomes much more frequent and decided, although in most cases less marked than in adult life. It is only as the age of puberty is approached that the lesions are practically the same as in the adult.

Typhoid bacilli are found in the various secretions and excretions, in different organs, the rose spots and the blood. They may be discovered in the stools often before the serum reaction can be obtained. The tissue-degenerations occurring in adult cases are absent or less marked in early life.

Symptoms.—The period of *incubation* is variable and difficult of precise determination. It may in general be placed at from 1 to 2 weeks. Languor, loss of appetite, and allied symptoms characterize it in the majority of cases. The actual beginning of *invasion* as marked by the development of fever which continues in average cases about a week before the second or *eruptive stage* is ushered in by the appearance of the roseola.

The symptoms of the attack vary according as the disease occurs in the new born, in infancy after this period, in early childhood (2 to 6 years), or in later childhood. These divisions are to be viewed merely as an artificial classification, since a great many exceptions exist, and subjects in one age-class may exhibit the symptoms of those pertaining to another. The peculiarities of the average case as observed in the latter part of early childhood and the first portion of later childhood may be reviewed as those characteristic of the general type for early life.

ORDINARY COURSE.—The principle symptoms distinguishing the disease in early life are: (1) The indefinite and uncharacteristic onset; (2) the shorter duration and greater mildness of the attack; (3) the disposition for nervous symptoms to over-balance intestinal ones. This does not mean at all that the nervous symptoms are actually more marked than in adult life.

In many cases the *onset* is very abrupt, particularly in young children, the disease becoming thoroughly developed in the course of a few days. Vomiting may be one of the first symptoms in such cases. The disease is occasionally ushered in by convulsions, or may begin violently with the manifestations of meningitis so well-marked that the true nature of the malady is discovered only later. In the majority of cases, however, the onset is peculiarly insidious and even long-continued, with symptoms so little pronounced that the roseola and enlarged spleen may, perhaps, be found upon the first examination. The child has been walking about with some degree of malaise, loss of appetite, slight headache and thirst, and with fever which has possibly not been recognized by the relatives. It is then difficult to determine just when the attack commenced. In other cases the initial stage is much shorter, the fever lasting only 2 or 3 days before the typhoid roseola appears.

When the disease has reached the second, or *eruptive stage* there is generally an absence of the severe nervous manifestations which constitute the typhoid state as seen in adult life; apathy and slight nocturnal delirium being the symptoms oftenest seen. The whole course of the attack is decidedly curtailed, especially the *stage of decline*. An average duration of the attack may be placed at from 14 to 20 days.

To this general description there are, of course, very numerous exceptions, and cases may vary in duration from that of the abortive type to that greatly prolonged; and in severity from the mildest to the most pronounced.

The symptoms can best be studied individually. Of the *gastro-intestinal* symptoms, vomiting is a frequent initial manifestation, and even later is more common than in adults, and sometimes almost or quite uncontrollable. I have seen this the direct cause of death. Sore throat is an occasional initial symptom and the tonsils may be red and swollen. The appetite is diminished and thirst is present. The tongue is generally coated, and often exhibits the red triangle at the tip and the red edges frequently described as characteristic of the disease. Dryness develops, as a rule, only in the severer cases in older children.

The condition of the bowels is variable. Constipation and diarrhea are about equally frequent, the former perhaps preponderating. Yet this depends upon the epidemic and there have been years in which nearly all the cases under my care had diarrhea, although generally not severe. The stools may show the typical pea-soup appearance. Involuntary evacuation occurs only exceptionally and in the severest cases.

The abdomen is generally only moderately distended and may be slightly tender in the right iliac fossa, but great distention and decided tenderness are much less often seen than in adult life.

Enlargement of the spleen is always present, although not always demonstrable. It can be detected in probably 80 to 90 per cent. of all cases in children. Russow¹ observed it in 85 per cent. of 1034 cases in children. It is discoverable at about the same time as the eruption. As



FIG. 93.—*TYPHOID FEVER WITH AN UNUSUAL DEGREE OF DEVELOPMENT OF THE ERUPTION.*

Minnie S., admitted to the Children's Hospital of Philadelphia Feb. 22, aged 5 years. Illness began 5 days before. Spots on abdomen observed the day before admission. The day after admission the spots were very numerous, and were seen also on the face. Spleen palpable. Recovery.

long as it persists the typhoid process cannot be considered completed. Lymphatic glandular enlargement is not infrequent but is generally slight. The typhoid *roscola* is sometimes stated to be less often present or less well developed in children. In my own experience this is not the case, and in 671 cases reported by Morse² the eruption was observed in 60 per cent. Henoch³ found it absent only 19 times in 381 cases. In average cases it is discoverable about the end of the 1st week. As in the adult, the spots appear in successive crops, each lasting about 3 days. Exceptionally the eruption is well marked over the whole body, involving even the face, and sometimes suggesting closely the early appearance of the rash of measles (Fig. 93). The spots continue to develop as long as the infection is active in the system. Sudamina are common, especially in older children. A branny desquamation during convalescence has been described as especially liable to occur in children. I have noted it frequently.

¹ *Padiatrija*, 1911, No. 3. Ref. *Monatsschr. f. Kinderh.*, Referat., 1912, III, 224.

² *Bost. Med. and Surg. Journ.*, 1896, CXXXIV 205.

³ *Kinderkrankheiten*, 1895, 773.

The *temperature* often runs a very irregular course. When typical it resembles that of adult cases except for the shorter duration and the frequent absence of the terminal remittent character (Fig. 94). In the initial stage it shows an evening rise and morning fall, with the gradual step-like ascent lasting until about the end of the 1st week. In very many cases, however, this stage is shortened greatly, and the fever rapidly rises to its maximum without the step-like character. In the second stage, that of the "acme," fastigium," or "eruptive period," the temperature remains continuously high, from 103° to over 104°F.,

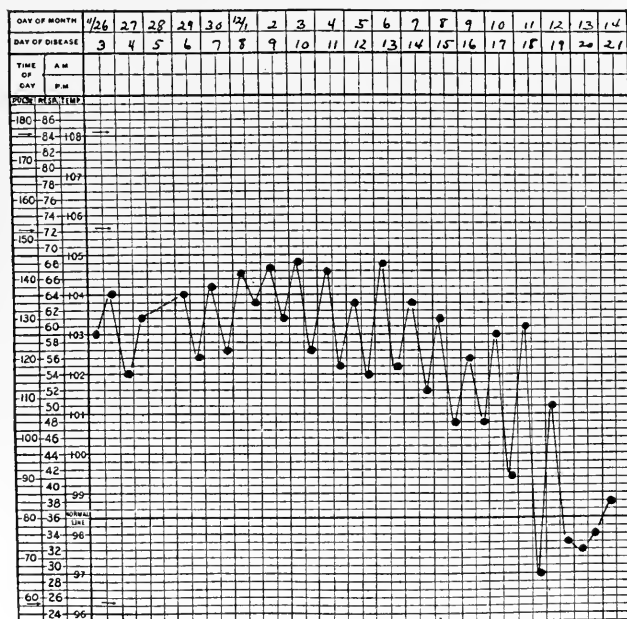


FIG. 94.—TYPHOID FEVER, AVERAGE CASE.

Evelyn P., aged 11 years. Nov. 26, chilly 2 days ago, been feeling tired, slight sore throat; Nov. 28, spleen enlarged to palpation, appetite poor; Nov. 30, general condition excellent, slight diarrhea, leucocytes 10,400; Dec. 2, rose spots found, Widal reaction negative; Dec. 6, mind entirely clear, slight diarrhea, Widal negative; Dec. 9, been no nervous symptoms except moderate apathy; Dec. 15, convalescent, appetite still poor.

(39.4° to 40°C.) with but little variation between morning and evening elevations. The high temperature, however, is generally much better tolerated than in adult life. In one instance a girl 10 years showed a temperature of 107°F. (41.7°C.) without the slightest sign of discomfort or of nervous symptoms. The temperature of the third stage, or stage of decline, is of much shorter duration than in adults (Fig. 95). According to Morse's statistics¹ the prolonged remittent form is absent in about $\frac{1}{2}$ of the cases. This absence doubtless depends on the lesser degree of intestinal involvement. The final fall is commonly much more rapid than in adults, and is often almost critical (Fig. 96).

Although the average duration of the fever is from 2 to 3 weeks, yet numerous exceptions are observed. Sometimes it lasts little over a

¹ Bost. Med. and Surg. Jour., 1896, CXXXIV, 205.

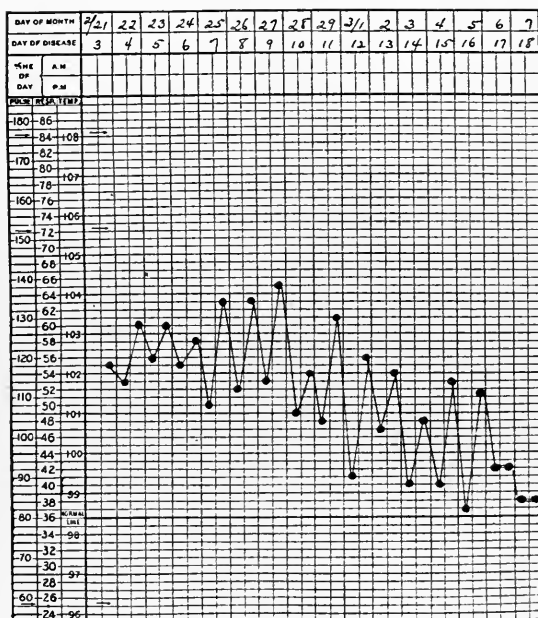


FIG. 95.—TYPHOID FEVER, ORDINARY FORM FOR CHILDREN.

Theresa F., aged 7 years. Came home from school with headache on Feb. 19; first seen Feb. 21; had not been in bed. During attack exhibited loss of appetite, enlarged spleen, very little apathy, Widal reaction. Was bright all the time. A few spots first found during a relapse.

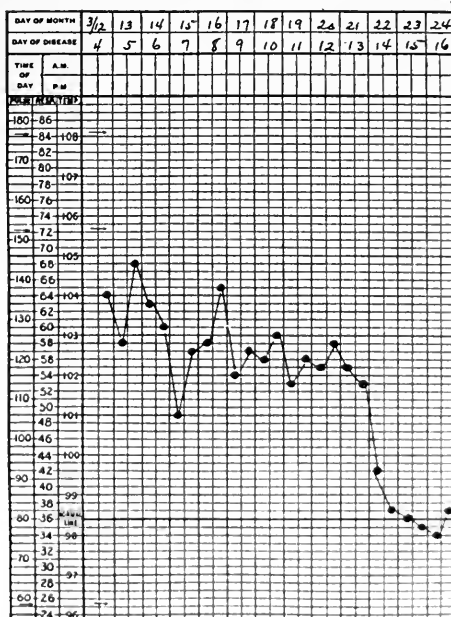


FIG. 96.—TYPHOID FEVER WITH CRITICAL FALL.

George C., aged 7½ years. Sudden onset with convulsion. During attack exhibited roseola, splenic enlargement, moderate diarrhea, good general condition. Temperature fell critically on the 14th day of the attack.

week, while in other cases it continues high with little change for 3 or 4 weeks or more. Before complete defervescence takes place there is shown a tendency for the temperature to become elevated from insignificant or undiscoverable causes (Fig. 97). This may in some cases result in an unusual prolongation of an irregular pyrexia. In other cases this is probably the result of an unusual persistence of the infectious process. In children apparently otherwise convalescent I have seen fever continue for 6 or 7 weeks (Fig. 98) and in one instance for over 12 weeks. How irregular and deceptive the course of the temperature may be at times is illustrated in the accompanying chart (Fig. 99) of a case in which only

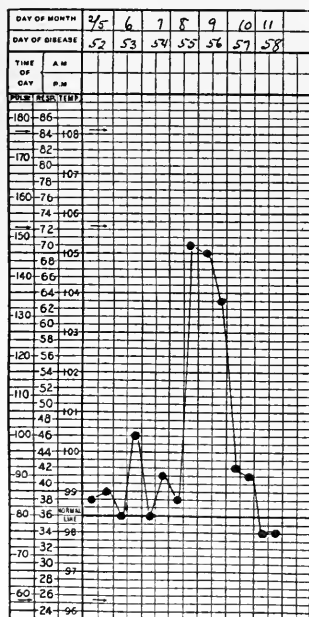


FIG. 97.

FIG. 97.—RECRUDESCENCE IN TYPHOID FEVER.

Chas. L., aged 5 years. Recovering from relapse.

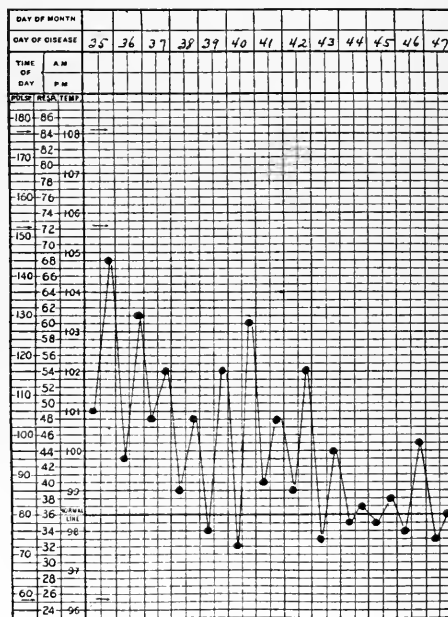


FIG. 98.

FIG. 98.—TYPHOID FEVER, PROTRACTED CASE, NO COMPLICATIONS.

Rose B., aged 8 years. Disease ran ordinary course, rather mild, splenic enlargement and rose spots. Then developed irregular fever without other symptoms or discoverable cause. This lasted until the 46th day.

the blood-culture made the suspected diagnosis finally certain. The chart does not represent the temperature-record at the same time daily, but rather the maximum and minimum for each day, there having been an entire absence of regularity in the hours of these occurrences.

The chief characteristic of the *blood* in typhoid fever is the absence of leucocytosis. The normal number of leucocytes belonging to the different ages of children (p. 59) must be taken into consideration in this connection, as also the very great ease with which many secondary conditions may produce leucocytosis in children with typhoid fever. An increase in the number of leucocytes is consequently not positive evidence that

typhoid fever is absent. A low leucocyte-count is of much more value in forming a diagnosis. Allowing for the influence of age, the differential count gives results identical with those seen in adults; there being a decrease in the number of neutrophils and an increase of the mononuclear cells, especially the lymphocytes. The eosinophils are diminished. The percentage of hemoglobin and of red cells is decidedly reduced as the disease advances. The typhoid bacilli may often be found in the blood before the Widal reaction can be obtained. After convalescence

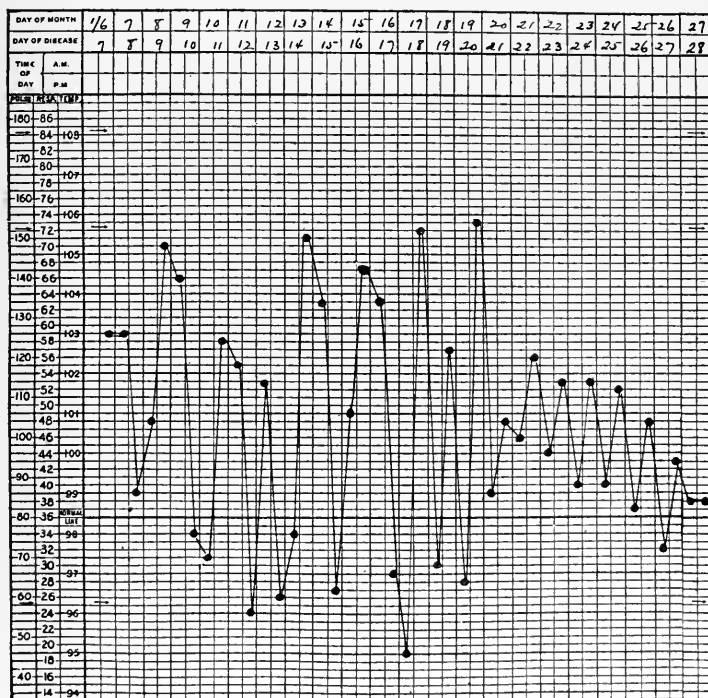


FIG. 99.—TYPHOID FEVER, UNUSUALLY IRREGULAR TEMPERATURES.

Christopher S., aged 8 years. Patient in the Children's Hospital, Phila. Jan. 7, intense headache, leucocytes at first 14,200, *tâche*, rigidity of neck, Kernig's sign, ankle-clonus, profuse sweating and chills, suspected of being malaria, cerebral abscess, meningitis, or sepsis, lumbar puncture negative, no plasmodia; Jan. 14, blood-culture positive for typhoid bacilli, rose spots, positive agglutinative reaction, leucocytes now 9800.

from severe attacks the hemoglobin especially shows a percentage-reduction. The blood-serum added to a fresh culture of typhoid bacilli causes a cessation of the movements and a clumping of the germs ("serum," "agglutinative," or "Widal" reaction). It occurs in about 95 per cent. of cases. It was present in 94 per cent. of 66 of my cases in the first 2½ years of life. It is usually not obtainable before the 5th or 6th day of the attack, or sometimes not until this is well advanced or the patient even convalescing. It may even be absent during the entire course of the primary attack and develop during a relapse. It may continue for months after the disease is over.

The *pulse*, as in adults, is not infrequently slower than the elevation of temperature would call for, and has a decided tendency to dirotism;

generally, however, only in older children. Not infrequently it is quite irregular and peculiarly slow during convalescence. In bad cases it may be irregular or unusually rapid during the course of the disease. The *arterial tension* is low. The cardiosphygmographic studies made by Schlieps¹ on 100 children with typhoid fever showed that the commonly observed arrhythmias are dependent upon sinus-irregularities and do not affect the prognosis. In severe attacks a dusky-red flushing of the cheeks sometimes indicates the vasomotor disturbance present.

Of symptoms connected with *respiration* cough is common yet seldom troublesome, and coarse râles are often heard in the chest. Epistaxis is probably not as common an initial symptom as in adults. Its occurrence in 40.9 per cent. of 550 cases as reported by Adams² would appear to be unusual. Except in the mild cases *emaciation* is commonly very decided and is sometimes extreme. *Nervous symptoms*, although more marked than intestinal, are, as stated generally less severe than in adult life. Headache is quite common, especially at the outset, but it is not often intense or persistent. In severe cases there may be much pain in the limbs, or the back or joints. I have known stiffness of the muscles of the neck to occur, without the mental symptoms which would suggest meningitis. The abdominal reflex is diminished. Many children remain in the best of spirits throughout the attack and do not feel or appear particularly ill; while others are usually irritable, especially if disturbed. Oftenest, however, the principal and the most characteristic nervous symptom is a decided apathy, with a tendency to be quiet and to sleep, which is increased when the temperature rises. Prostration is seldom as great as in adult cases, except after unusually severe attacks or in patients in whom some debilitating cause, such as severe diarrhea, has been present. Delirium is generally absent or slight, occurring only at night, but in severe cases may become very pronounced. Convulsions sometimes usher in the attack (Fig. 96) even in cases which are not later severe. Coma, stupor, coma vigil and subsultus are rarely seen except in older children. A condition strongly suggesting meningitis is sometimes observed during much of the attack. This is not frequent, although probably oftener seen than in adults (Fig. 106).

The *urine* exhibits a decided diazo reaction. Febrile albuminuria may be present if the temperature is high, but evidences of nephritis are not as common as in adults. Acetone is occasionally found.

VARIATIONS IN TYPE.—Different types of typhoid fever seen in early life may be described. The classification may be made either (*A*) according to the age of the patient, or (*B*) according to the characteristics of the attack in general.

(*A*) Based upon *age* the following types may be mentioned:

1. Fetal and Congenital Typhoid Fever.—The only distinction between these two varieties is that in the latter the child is born alive and consequently exhibits symptoms.

The majority of pregnant women suffering from typhoid fever abort. The collected statistics of Etienne³ show this occurrence in 70 per cent. of pregnant women with the disease. Nothing in the superficial appearance of the body of the infected fetus indicates the nature of the disorder, but the germs may be recovered from the blood and the organs. In

¹ Jahrb. f. Kinderh., 1911, LXXIV, 386.

² Amer. Journ. Med. Sci., 1910, May.

³ Gaz. hebdom., 1896, XLIII, 184.

the congenital cases the infant is born alive, sometimes prematurely and sometimes at term. Life may continue only a few minutes, the cause of death not being apparent. In others the disease may last several days, or even 2 weeks or longer. The symptoms are usually entirely uncharacteristic. Fever is generally present; and convulsions, jaundice diarrhea, constipation, tympanities, roseola, enlarged spleen, cough, vomiting, intestinal hemorrhage and purpura have been reported. Death almost always occurs. As already pointed out the discovery of the Widal reaction in these cases cannot be looked upon as diagnostic, since it may be found in apparently healthy children born of typhoid mothers, the agglutinating principle having entered either by way of the placental circulation or through the milk. (See p. 391.)

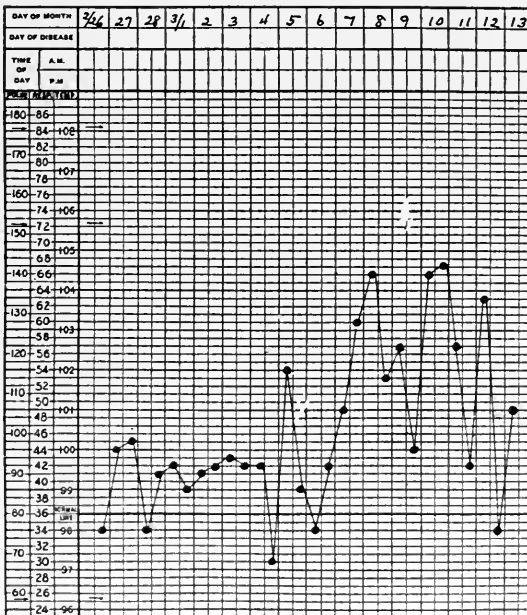


FIG. 100.—INFANTILE TYPHOID FEVER.

William H., aged 3 months. Child restless, with slight cough, abdominal distention, moderate diarrhea with greenish stools, increasing weakness, stupor and loss of appetite. Death on Feb. 13. Diagnosis supposed to be ileocolitis. Autopsy showed spleen soft and much enlarged, Peyer's patches and mesenteric glands hyperplastic, Widal reaction in heart's blood. Case illustrates severe course with uncharacteristic symptoms.

2. Infantile Typhoid Fever.—Under this heading may be included cases in which the affection was acquired after birth and during the first 2 years of life. The disease in this period, and especially in the 1st year, is often marked by the absence of most of the characteristic symptoms, the diagnosis being impossible until the typhoid eruption or the agglutinative reaction is discovered. The temperature is generally high and very irregular in type, and is usually attributed to some of the many other causes of fever in early life. Very often the disease is supposed to be an ileocolitis of moderate severity, since diarrhea is a frequent symptom common to both (Figs. 100, 107, 109). Diarrhea, tympanites and vomiting are rather more frequent at this time of life than later.

Bronchitis is of common occurrence; epistaxis uncommon. The patient is often prostrated, and may seem more ill than the symptoms rationally explain. The pulse is rapid and nervous symptoms may be marked, fretfulness and restlessness being more frequently seen than apathy. As in fetal typhoid the disease often takes the form of a blood-infection without special local symptoms (Fig. 107).

3. Typhoid Fever in Early Childhood.—At this period, from the age of 2 up to that of 6 years, the attack is usually of a more benign type than at any other time of life. It is now that the characteristics already described as those of the disease in early life are most prone to show themselves. Diarrhea is less common than either in infancy or in later

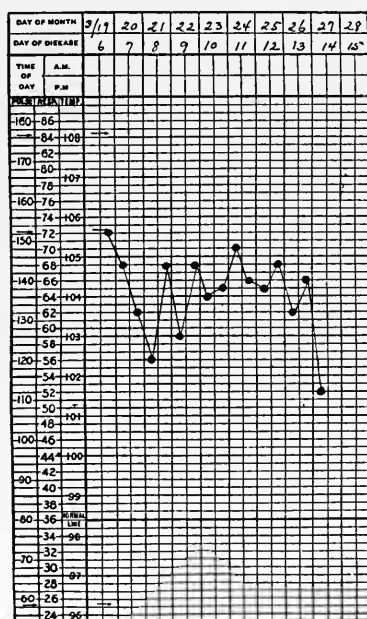


FIG. 101.—TYPHOID FEVER IN EARLY CHILDHOOD, SEVERE CASE.

Sadie T., aged 3 years. Rapid onset with fever and prostration and loss of appetite. During attack suffered from slight diarrhea, numerous râles in chest, occasionally dyspnea, apathy, sopor, sometimes great irritability, occasional vomiting, severe abdominal distention, cyanosis. Death on the 14th day of the disease.

childhood and is seldom troublesome. The temperature is more suggestive of typhoid fever than in infancy, but that of the third stage is generally much abbreviated and not of a remittent type. The course is short. Nervous symptoms are nearly always mild except in the cases with a meningitic onset. Complications are not as frequent as later. To this description there are of course numerous exceptions, and the attack may be very severe (Fig. 101).

4. Typhoid Fever in Later Childhood.—This type is distinctly more like that seen in adults, and the nearer the child is to puberty, the closer is the resemblance liable to be. This is especially true after the age of 10 years (Fig. 102). Diarrhea may now be troublesome, and hemorrhage and perforation are more liable to occur than earlier, all this depending upon the greater likelihood of intestinal ulceration at this time

of life. The symptoms of the typhoid state are more prone to develop, but only in severe cases to any great degree. The course is apt to be longer than in early childhood, and the fever of the third stage is oftener of the remittent type.

(B) Based also upon the *symptoms* as a whole, regardless of age, the following varieties may be mentioned:

1. Abortive Form.—In this form all the characteristic early symptoms may be present in a mild (Fig. 103) or in the ordinary or a severe

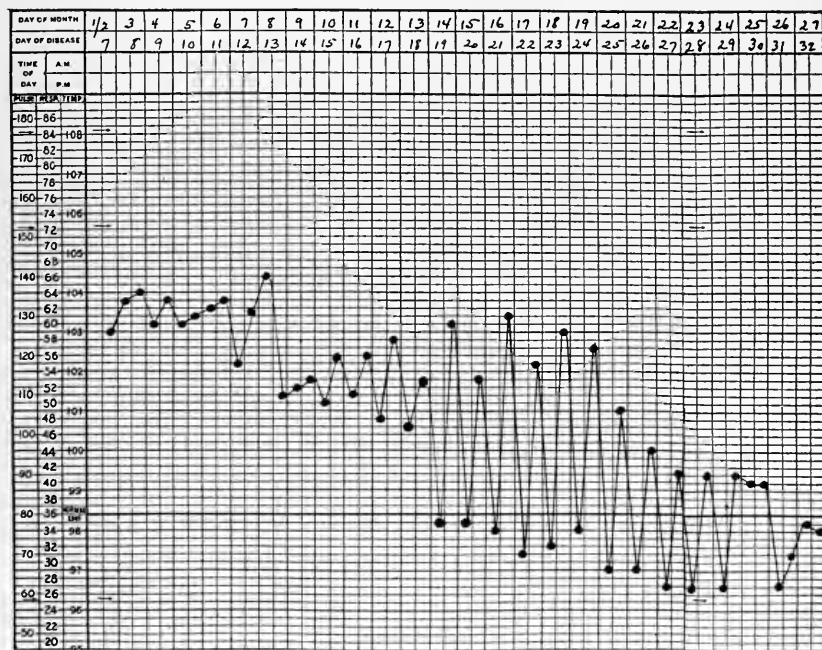


FIG. 102.—TYPHOID FEVER, LATER CHILDHOOD. SEVERE CASE OF ADULT TYPE OF DISEASE.

Thelma M., aged 11 years. Suffered from headache, stuporous most of time, muttering delirium, carphologia, occasionally actively delirious, coated tongue, dry lips, troublesome abdominal distention.

form (Fig. 104), but the course is greatly abbreviated, lasting only 8 to 10 days. These cases are not infrequent, being much more common than in adults.

2. Mild Form.—This variety may be abortive also, as regards its duration, but the term is much better applied to those cases in which all the symptoms are peculiarly mild although the attack is not unusually curtailed. It is the form very commonly seen in early childhood. There is no diarrhea, loss of appetite, or prostration; and, in fact, practically no subjective symptoms except possibly slight headache and a trifling degree of apathy. Only the continued fever of moderate degree, the Widal reaction, and possibly the discovery of rose spots and enlargement of the spleen indicate the presence of the disease. Even the temperature may in occasional cases scarcely exceed 100°F. (37.8°C.) (*Afebrile form*). Sometimes only the occurrence of the case in a family outbreak makes the diagnosis clear (Fig. 105).

3. Nervous Form.—This occurs not infrequently, and is often the cause of great difficulty in diagnosis. The disease may begin abruptly as a pseudomeningitis, with repeated convulsions continuing several days; or there may be unconsciousness, grinding of the teeth, intense restlessness, rigidity of the neck, and possibly an unusually active delirium. These early symptoms may soon give place to the more usual ones of the disease; but sometimes, particularly in older children, severe nervous symptoms may not develop until later (*Meningitic typhoid*) (Fig. 106).

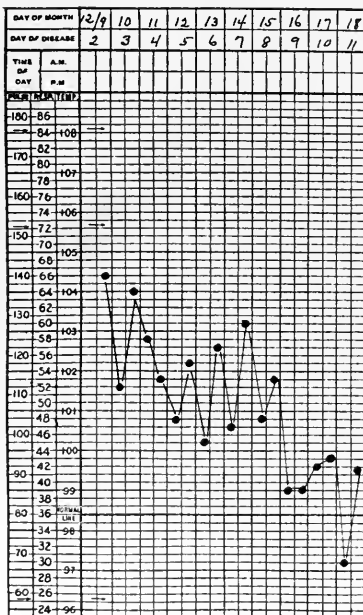


FIG. 103.

FIG. 103.—ABORTIVE TYPHOID FEVER, COURSE SHORT AND SYMPTOMS MILD FROM BEGINNING.

Thelma M., aged 11 years. Onset sudden. Symptoms consisted solely of slight weakness and diffused aching, fever, splenic enlargement. Fever ceased on 9th day and child out of bed on 11th day of disease. Case supposed to be influenza until a positive Widal reaction and later a very severe relapse (Fig. 109) showed the nature of the original attack.

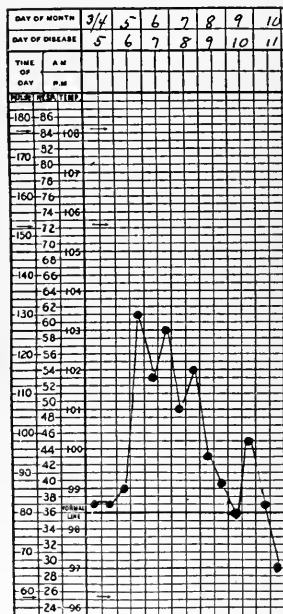


FIG. 104.

FIG. 104.—ABORTIVE TYPHOID FEVER, SEVERE INITIAL SYMPTOMS.

Annie A., aged 7½ years. Symptoms of invasion consisted of fever, very frequent vomiting, diarrhea. Then improved, felt perfectly well and was bright, no apathy, vomiting and diarrhea ceased. Enlargement of spleen and roseola (?) present. Widal reaction positive. No fever after 10th day of disease.

Complications and Sequels.—Complications and sequels are not common in average cases in children. Those of the *respiratory tract* are perhaps most frequent. Bronchitis is common, especially in the severer cases of the disease. Pneumonia occurs, although perhaps less often than in adults. Severe laryngitis is an occasional complication. I have known a child to be aphonic for 5 or 6 weeks. In rare instances ulceration with stenosis may occur and intubation or tracheotomy be required. In 1 instance a tracheotomy tube was still being worn after 3 years.

Some of the cases of laryngeal involvement depend upon a laryngeal perichondritis. I have observed this in a few instances. Pleurisy is unusual. Abscess and gangrene of the lung have both been reported, and hypostatic congestion may occur in severe cases. Disorders of the *circulatory apparatus* are for the most part infrequent. Pericarditis and endocarditis are rare, venous or arterial thrombosis very exceptional. More or less anemia is a natural sequel in severe cases. A hemorrhagic tendency with the production of purpuric eruptions, bleeding of the gums and other evidences of the hemorrhagic diathesis, is very uncommon at any time of life (*Hemorrhagic typhoid*). Epistaxis may be so severe that it

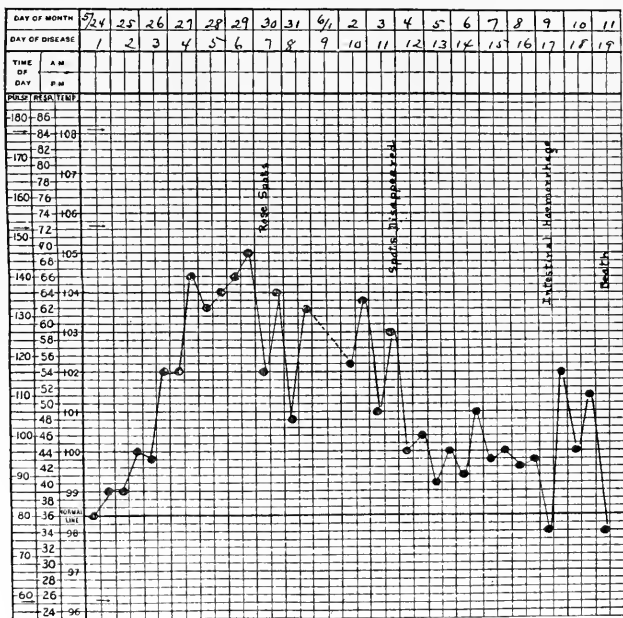


FIG. 107.—TYPHOID FEVER WITH FATAL INTESTINAL HEMORRHAGE.

Martin D., aged 5 months. Case shows short duration of 1st and 3d stages as well as of the attack as a whole. Case supposed at first probably to be pneumonia, although there were no positive symptoms of any sort. Chart shows temperature from the day of onset. Child suffered from restlessness, crying, loss of appetite, diarrhea, vomiting, later rose spots and enlarged spleen. Improved rapidly then developed intestinal hemorrhage with return of vomiting, abdominal pain and tenderness, feeble circulation, death. Autopsy. Later found that the mother also was in the Philadelphia Hospital with typhoid fever.

may be ranked as a complication. I have seen it the direct cause of death. Disturbances of the *digestive system* are important. Parotitis is only occasionally seen, but generally proceeds to suppuration (p. 668, Fig. 233). Severe aphthous stomatitis is sometimes observed, or ulcerative stomatitis going on to necrosis of a small portion of the bone with loss of teeth. Pseudo-membranous tonsillitis occasionally occurs. Noma, although uncommon, is more liable to develop after typhoid fever than after any other disease except measles. Severe diarrhea is not infrequent in later childhood. Fecal impaction is an unusual complication, of which I have seen but 1 instance. Intestinal hemorrhage is rare as compared with adult life, and is met with almost only in children of 10 years or

older. Only 9 cases; *i.e.* 1.6 per cent. of 553 cases of typhoid fever in children collected by Morse,¹ suffered from this symptom. I have, however, observed it in many more cases than this, in 1 instance in an infant of 5 months (Fig. 107). Costinesco² reported it 22 times in 762 cases of typhoid fever in children. It has been witnessed in some of the congenital cases, but is then rather a manifestation of a general hemorrhagic state dependent upon the septic condition of the blood. Intestinal perforation is another unusual complication in early life. Henoeh³ saw it but

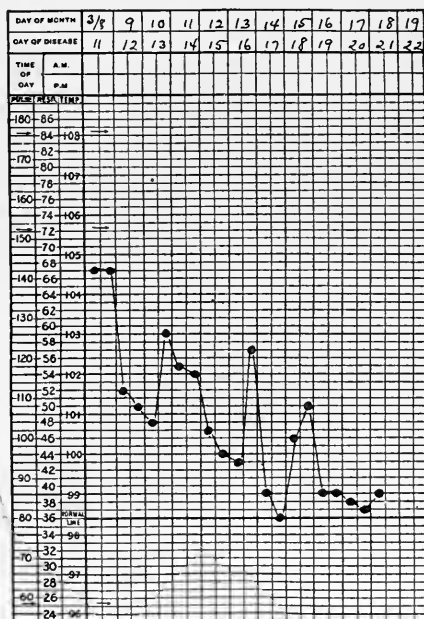


FIG. 108.—TYPHOID FEVER WITH INTESTINAL PERFORATION, ILLUSTRATING ABSENCE OF CHARACTERISTIC SYMPTOMS OF PERFORATION AS OFTEN SEEN.

Mary B., aged 4 years. Admitted to the Children's Hospital Mar. 8. Said to have had slight symptoms of typhoid fever for 10 days. Attack ran a mild course, although with considerable abdominal distention. Vomited twice in the morning of the 18th and twice in the afternoon. Had occasional slight abdominal pain at first, no distention or tenderness at any time. Sat up in bed through the morning and did not look ill. In the afternoon looked more ill and temperature had risen to 105.6° F. by 6 p. m. Strength now failed rapidly, but entirely without abdominal symptoms. Died 6 a. m. on the 19th. Autopsy showed perforation.

once in 381 cases, Morse⁴ not at all in 284 cases, Rennert⁵ 4 times in 471 cases, and Schulz⁶ but 8 times in children. It is, however, not so rare as often supposed. Montmollin⁷ reported it 7 times in 90 cases in children, Adams⁸ in 11 of 337 cases and Setbon⁹ 26 times in 1506 collected

¹ Bost. Med. and Surg. Journ., 1886, CXXXIV, 205.

² Thèse de Paris, 1897, 35.

³ Kinderkrankheiten, 1895, 770.

⁴ *Loc. cit.*

⁵ Deutsch. med. Wochens., 1889, 1063.

⁶ Jahrb. d. Hamburger Stadts Krankenanstalten, 1889, 1, 7.

⁷ Thèse Neuchâtel, 1885.

⁸ Arch. of Pediat., 1904, XXI, 81.

⁹ Thèse de Paris, 1902.

cases. Out of 289 cases of operation for typhoidal perforation collected by Elsberg¹ 25 occurred in children less than 15 years of age, and Jopson and Gittings² add 45 reported cases to this list. I have personally observed 9 instances, 6 of these cases previously reported³ 1 being a girl of 4 years and another of 6 years. Generally, however, it occurs only in patients near the end of later childhood. It is to be noted that the symptoms of perforation in early life are not infrequently much less marked than in adults and very misleading, and that the diagnosis is often extremely difficult. The usual fall of temperature with symptoms of collapse and severe abdominal pain may entirely fail to develop. This occurs more frequently than is the case in adults (Fig. 108). Typhoidal cholecystitis is probably of much more common occurrence than formerly supposed. It may rarely exhibit itself in an acute form with perforation and secondary peritonitis, as in cases reported by Bittner.⁴

Among *nervous complications* and sequels a temporary aphasia is more liable to occur in early life than later. Henoch⁵ observed this in 20 of his 381 cases. I have seen it complete for several weeks. The children sometimes appear to be suffering not so much from inability to express their thoughts in words, as from a dullness of mind which has removed the desire. Post-typhoidal insanity is a sequel decidedly rare in children. Adams⁶ among others, reported 4 cases, and I have observed it a number of times. Chorea is a not infrequent sequel. Meningitis is a very unusual complication, the great majority of the cases showing symptoms of this condition being in reality instances of pseudomeningitis. Nevertheless, undoubted typhoidal meningitis may occur and the typhoid bacilli have been recovered from the fluid obtained by lumbar puncture or from the meninges. Paralysis resulting from neuritis has been reported, and rarely a hemiplegia of cerebral origin. *Otitis* is a complication occurring much more frequently than in adults. It is often non-purulent, producing decided but temporary deafness; often purulent with consecutive perforation. Sometimes the deafness appears to depend on a central disturbance rather than upon otitis. Purulent otitis was reported in 2.7 per cent. of Adams⁷ cases.

Affections of the *genito-urinary apparatus* may occur. Nephritis is an occasional complication much less often seen than in adults. It generally recovers as convalescence from the typhoid fever proceeds. Cystitis and pyelitis are not common sequels in early life. *Suppurative processes* in various regions may take place. One of the most troublesome forms is furunculosis, which may be very extensive and severe. Subcutaneous abscesses are often observed. Bed-sores are very much less frequent than in adults and are observed only in neglected cases in older children. Suppuration of the mesenteric glands may occur, and cases have been reported in which the symptoms strongly suggested intestinal perforation (Rowland).⁸ Suppuration of the joints or bones and necrosis of the bones are occasional sequels. The typhoid spine may sometimes be observed in early life, though less often than in adults.

Cutaneous eruptions sometimes occur as complications. A rubeoloid

¹ Ann. of Surg., 1903, XXXVIII, 71.

² Amer. Journ. Med. Sci., 1909, CXXXVIII, 625.

³ Amer. Journ. Med. Sci., 1905, Oct.

⁴ Prag. med. Woch., 1914, XXXIII, 279.

⁵ Kinderkrankheiten, 1895, 771.

⁶ Trans. Amer. Ped. Soc., VIII, 177.

⁷ Amer. Journ. Med. Sci., 1910, May.

⁸ Journ. Amer. Med. Assoc., 1906, XLVI, 507.

and, less often, a scarlatiniform rash are occasionally seen, and sometimes urticaria. These may be present either early or later in the attack. Herpes is rare, but probably more common than in adult life. Other *acute infectious diseases* may complicate typhoid fever or immediately precede or follow it. This is true, for instance, of scarlet fever, measles, rubella, varicella, pertussis, diphtheria, cerebrospinal fever, malaria, and erysipelas. The occurrence of a secondary diphtheria seems to be especially frequent.

Relapse.—Relapse certainly is not less, and perhaps more, frequent than in adult life. Koplik and Heiman¹ in an analysis of 160 cases of

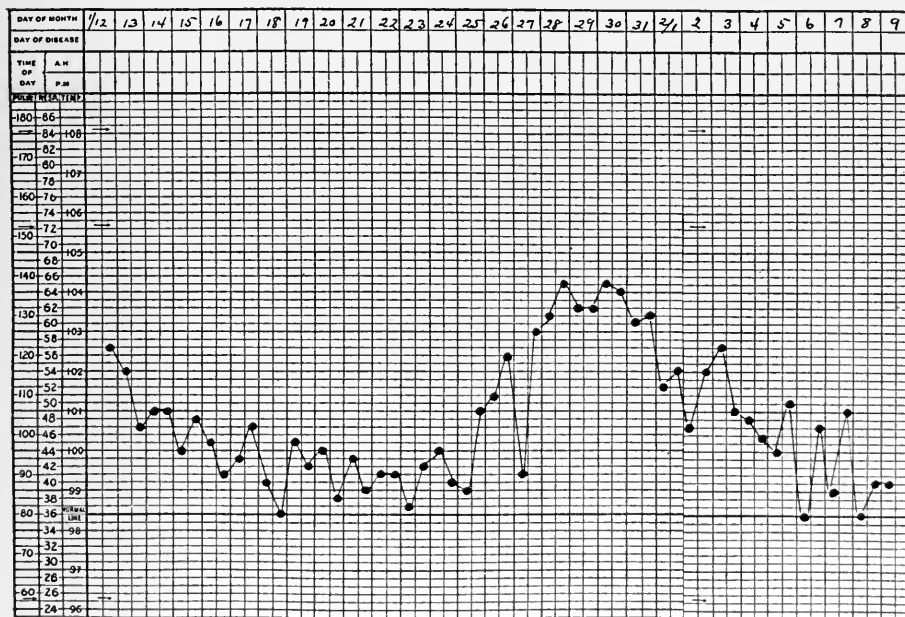


FIG. 109.—RELAPSE IN TYPHOID FEVER.

Irene T., aged 9 months. Showing relapse with return of fever, roseola, and splenic enlargement.

typhoid fever in children reported relapse in 15 per cent.; and Adams,² in 550 cases, 8.7 per cent. with relapse. In my series of 75 cases in the first 2½ years of life there were 3 undoubted instances of relapse. It develops even as early as the 3d or 4th, but oftenest from the 14th to the 17th, day after the temperature of the first attack has reached normal. Relapse must, of course, be sharply distinguished from the *recrudescence* of fever which lasts but a day or two and is brought about by many slight causes (Fig. 97). It is characterized by a return of the usual symptoms, including the reappearance of the roseola and of the enlargement of the spleen. Not uncommonly some degree of splenic enlargement persists during the afebrile interval. The relapse may equal, exceed, or fall below the first attack in duration and severity. It is generally of shorter duration. The age of the child seems to exert no

¹ Arch. of Ped., 1907, XXIV, 1.

² Amer. Journ. Med. Sci., 1910, CXXXIX, 638.

decided influence on the tendency to its development. In 1 instance it occurred in a female infant of 9 months (Fig. 109). Even more than one relapse may be observed. In 1 case under observation 3 relapses were witnessed; and David¹ reported 5 relapses in a boy of 11 years.

Recurrence.—One attack of typhoid fever usually confers lasting immunity. This is, however, by no means so generally true as in the case of scarlet fever and measles. The immunity may be only a temporary one. Even 3 or 4 attacks of typhoid fever may rarely occur in the same person.

Prognosis.—The mortality of typhoid fever in early life is, on the whole, less than later. In the 192 cases in children reported by Schavoir² only 2 died, a mortality of 1 per cent. This is exceptionally low. In Morse's³ 284 cases the mortality was 6 per cent. against that of 13.5 per cent. in 3396 adult cases. Of 432 cases occurring in the Children's Hospital of Philadelphia during 14 years 23; *i.e.* 5.32 per cent., died. In general the mortality may be placed at from 4 to 5 per cent., yet this varies very much with the *age*. In infancy the number of fatal cases is large. Of 278 collected cases⁴ not over $2\frac{1}{2}$ years of age 57 per cent. died. Although this figure is exceptionally high, due probably to the fact that very many of the milder cases were not considered worthy of being reported, and is not to be considered representative, it serves to show that the disease at this period is much more fatal than later. In the later reported⁵ 75 cases under $2\frac{1}{2}$ years the mortality was 12 per cent. In early childhood the mortality is at its lowest and probably does not exceed from 2 to 4 per cent. It increases steadily as the age of puberty is approached. The lesser mortality of early life depends in part upon the lesser severity of the disease, and in part upon the lesser frequency of dangerous complications and sequels.

Among *unfavorable prognostic symptoms* are very severe diarrhea; obstinate vomiting; persistent dryness of the tongue; a marked degree of tympany; a weak, rapid pulse; and the development of unusual degrees of stuporous mental states or other nervous phenomena. The disappearance of the diazo-reaction is claimed to be a favorable symptom, indicating that a fall of temperature will occur in a few days (Rolleston).⁶ The slow, irregular pulse of convalescence is not an unfavorable symptom.

Diagnosis.—This offers many difficulties in early life, especially in infancy. In typical cases the continued fever with no other discoverable cause, the enlargement of the spleen, the rose spots, the agglutinative reaction, the absence of leucocytosis, the discovery of typhoid bacilli in the feces, urine and blood, and the occurrence sometimes in family epidemics serve to render the diagnosis easy, although it may be days before any conclusion can be reached. Most important is the serum reaction, and it is upon this that the diagnosis must rest in many obscure cases. Yet in many instances in which other symptoms are positive, the serum reaction can at no time be obtained. This does not militate against the correctness of the diagnosis.

The absence of leucocytosis is often a valuable diagnostic sign, but the presence of this condition does not exclude typhoid fever. The diazo-

¹ Zentralbl. f. inn. Med., 1912, XXXIII, 1071.

² Med. Rec., 1895, XLVIII, 803.

³ Bost. Med. and Surg. Journ., 1896, CXXXIV, 205.

⁴ Griffith and Ostheimer, Amer. Journ. Med. Sci., 1902, Nov.

⁵ Arch. of Pediat., 1912, Aug.

⁶ Lancet, 1905, I, 290.

reaction is only a corroborative indication since it may occur in some other infectious fevers. Continued temperature without evident sufficient cause is always suspicious, but as this may develop also in acute miliary tuberculosis, grippe, ileocolitis, low-grade bronchopneumonia, and other conditions, it cannot be regarded as a safe diagnostic symptom.

Typhoid fever is liable to be confounded in early life with a number of other affections. First among these, especially in infancy, is *ileocolitis*. Diarrhea is quite common in typhoid fever at this period of life and tympanites may occur in either disease. The intestinal condition in ileocolitis is, however, generally much more severe and out of proportion to the degree of fever and a leucocytosis is generally present. Later the discovery of rose spots with decided enlargement of the spleen and a positive agglutinative reaction may make the diagnosis of typhoid fever clear.

Malaria of a continued febrile type may simulate typhoid fever very closely. Leucocytosis is absent in each, and enlargement of the spleen present. The discovery of the plasmodium and the absence of the Widal and diazo-reactions serve to distinguish malaria. The course of the temperature, with regular intermissions or remissions, is likewise characteristic.

Grippe is, at times, especially in the early stage, readily supposed to be typhoid fever. Later the short course of the attack and the development of characteristic symptoms serve to distinguish it. There are not infrequently instances, however, in which the fever of grippe is unusually prolonged, the prostration decided, and the symptoms little characteristic, and these cases may cause much perplexity. Leucocytosis may be absent in grippe and enlargement of the spleen present in both diseases. The discovery of the Widal reaction and of rose spots will settle the diagnosis.

Acute miliary tuberculosis in many instances resembles typhoid fever closely. It exhibits a fever of the continued type, the absence of leucocytosis, and the presence of splenic enlargement without any discoverable cause or any localization of the tuberculous process. The subcutaneous tuberculin reaction cannot be sought for on account of the continued presence of fever, and the cutaneous reaction is not to be depended upon in this condition. The persistent failure of the Widal reaction and of rose spots to appear, the continuance of the pyrexia beyond the duration of that of typhoid fever, and the possible development later of localizing symptoms in the brain or the lungs may finally serve to distinguish tuberculosis. Sometimes a decided dyspnea without discoverable pulmonary lesions to account for it is present in miliary tuberculosis. Occasionally tubercles may be discovered in the choroid. Often however, the diagnosis cannot be made during life.

A *continued fever of intestinal origin*, dependent probably on a mild toxemia the result of chronic digestive disturbances, often occasions difficulty in diagnosis, especially in the first 2 years of life. Fever of this nature is liable to be of a very irregular type, and moderate diarrhea is often, although not always, present. Only careful watching of the course of the case and the continued absence of the rose spots, enlarged spleen and Widal reaction serve to exclude the presence of typhoid fever.

Meningitis may at first resemble typhoid fever. The vomiting and apathy often seen in the latter may suggest tuberculous meningitis, while the cases of typhoid fever of the meningitic type, beginning with convulsions and with severe cerebral symptoms, may point to cerebro-

spinal fever. In tuberculous meningitis, however, there is a tendency to leucocytosis, and in cerebrospinal fever the leucocytes are very much increased in number. The diazo-reaction is present in both forms of meningitis as well as in typhoid. The meningitic symptoms of typhoid fever, however, generally occur early and disappear soon. They consist usually, too, of symptoms of excitement, and rarely in children assume the form of paralysis and coma. Kernig's sign is of little value as a differential symptom. In doubtful cases the continued absence of the Widal reaction and the results of lumbar puncture serve to distinguish meningitis.

Treatment. Prophylaxis.—The prevention of the direct spread of the disease from the patient is to be sought by careful disinfection of the urine and feces and of all the bed and body linen; the linen by submerging in a 5 per cent. carbolic acid solution and afterward by boiling; the excretions by mingling them with this solution or with equal parts of chloride of lime and water. The hands of the attendants should be washed and disinfected after handling the patient.

The prevention of extension in general is, of course, to be accomplished chiefly by the employment of water which is entirely above suspicion. When this cannot be obtained all water to be used for drinking and bathing, and for the washing of vegetables, fruits, eating utensils, nursing bottles and nipples, and the like, should be boiled. The systematic heating of milk to a temperature of 60°C. (140°F.) for 5 minutes will destroy any typhoid germs present.

Immunizing Treatment.—Experimental work has been done in this line by Wright,¹ Pfeiffer and Kolle² and others who employed injections of cultures in which the germs had been killed by heat. The results reported in the United States Army by Russell,³ Lyster⁴ and others have been and continue to be most encouraging.

Treatment of the Attack.—This is purely expectant and symptomatic; the matter of the greatest importance being that of avoiding an excess of it. No specific treatment has yet been proven certainly effective, although the results obtained encourage further trial. Wright⁵ employed inoculations with dead bacteria, Chantemesse⁶ and Josias⁷ injections of an immunizing serum, and Jež,⁸ a liquid prepared from the organs of immunized animals and given by the mouth. Good results are reported by all. Favorable results with the serum in modifying the course of the attack in the case of children have later been reported by Josias;⁹ and with the vaccine by Ortiz, Acuña and Belloc.¹⁰ Treatment directed to the disinfection of the intestinal tract has not proven to be of any special value. This has been at least the experience of many observers as well as my own.

No matter how well the child feels, confinement to bed is imperative. This need not be continued as long after defervescence as is commonly required with adults. The diet should be easily digestible but abundant, one in which milk forms a prominent part being the best in

¹ Lancet, 1901, II, 1107.

² Zeitschr. f. Hyg., 1896, XXI, 203.

³ Journ. Amer. Med. Assoc., 1914, LXII, 1371.

⁴ Journ. Amer. Med. Assoc., 1915, LXV, 510.

⁵ Lancet, 1901, I, 339.

⁶ Annals inst. Pasteur, 1892, VI, 755.

⁷ Ann. de méd. et chir. inf., 1903, XI, 438.

⁸ Wien med. Wochenschr., 1899, XLIX, 346.

⁹ Acad. de méd., 1906, March 6. Ref., Arch. f. Kinderh., 1908, XLVII, 454.

¹⁰ Arch. de méd. des enf., 1915, XVIII, 575.

most cases. There is no necessity, however, of making milk the only food, and a regimen may well be used in which there is a high carbohydrate percentage in the form of gruels and the like. The fancies of the patient are to be humored as far as possible, since, although over-feeding is to be avoided, it is certain also that many patients are under-fed. Drinking water should be offered freely and often. As convalescence begins the appetite returns, and the diet should be decidedly increased in quantity and variety. This may usually be done earlier than has been the custom in adult cases, owing to the lesser degree of intestinal involvement present. The increase must, however, be made cautiously, since disturbance of digestion appears undoubtedly to favor the development of relapse, and certainly can cause a recrudescence of fever.

The surface of the body should be kept clean by sponging with water or alcohol and water, and the mouth should be washed with a solution of boric acid several times a day. The need of the patient for undisturbed sleep must be emphasized. It is well at night to lengthen decidedly the intervals between the administerings of medicines or other treatment, and to forsake regularity, utilizing the times when the patient wakes. Of course in very severe cases this suggestion is not applicable.

Treatment of special symptoms may next be considered. Of the *temperature* it is well to remember that children both attain decided elevation oftener and endure it better than do adults. Consequently mere elevation, if not prolonged and if unattended by unfavorable symptoms, need not cause alarm, and does not require treatment. In fact the chief object of hydrotherapy is not so much the reduction of temperature as the stimulating effect upon the circulation and the controlling of nervous manifestations. Should a reduction seem indicated, either sponging or tubbing may be employed. Where there is active objection on the part of the child to the use of water it will be found that tubbing is not only more effective than sponging but much less troublesome both to the nurse and to the patient, since it requires a shorter time and causes no more, or less, opposition. Sponging, to have any antipyretic value, must be kept up for from 10 to 20 minutes and repeated frequently. Often the cold or warm pack is very efficient and disturbs the child but little. Cold tub-baths are, however, almost never required and children bear them badly. A graduated bath of 95°F. (35°C.) reduced by the addition of cold water to 85°F. (29.4°C.) is that most often serviceable. It may be given every 3 hours when the temperature exceeds 103°F. (39.4°C.), and continued from 5 to 10 minutes according to the effect desired and the tolerance shown. The child while in the bath should be rubbed vigorously and briskly as a stimulant to the circulation. The head should, meanwhile, be kept cool by the repeated application of cloths dipped in cold water.

Very often even the graduated bath causes a greater degree of cyanosis and of weakness of the pulse than can be considered safe. In such cases the warm tub bath of 95° to 100°F. (35° to 37.8°C.) is frequently very efficacious. Yet not uncommonly even this is followed by imperfect reaction and should be abandoned. In other cases the intense excitement and opposition shown when a bath is given produces injurious fatigue. In fine, hydrotherapy, while a most valuable agent in typhoid fever, must never be employed in early life as a routine measure without a close study of its effects upon the individual case; otherwise more harm than good may result. If there is much shivering and blueness after the bath an alcoholic stimulant should be administered. Frequently it is advisable

to precede it by this also. The temperature of the patient may be taken half an hour after the bath is over, to determine what degree of lowering has taken place.

The tub bath is contraindicated in cases of intestinal hemorrhage and in subjects extremely prostrated, where moving would be too exhausting. In some cases a satisfactory reduction of temperature may be obtained by the nearly constant application of an ice-bag to the abdomen, with one or more layers of towel intervening. In infants ice must always be employed guardedly, as it is not always well tolerated. It is only exceptionally, where hydrotherapy cannot be employed and where it is desired to reduce unusually high temperature, that the cautious administration of coal-tar antipyretic drugs may be required. In the cases where the terminal stage of typhoid fever is unusually prolonged, or where an irregular temperature persists from causes entirely undiscoverable, the continued employment of fairly large doses of quinine is occasionally very serviceable. Sometimes these patients are finally most benefited by getting them out of bed into a chair. I have often seen apyrexia promptly follow this procedure.

Prostration demands stimulating treatment, especially by alcohol. While the use of alcohol, as of stimulants of any sort, is probably not a necessity in the majority of cases of typhoid fever, yet in small amounts it is often, I think, an excellent conservator of energy. The condition of the heart-strength is the chief guide. Any degree of prostration, weakness of pulse, nervous exhaustion, or impairment of the first sound of the heart necessitates stimulation by this or by other drugs. It is easier to maintain a fair condition of the pulse by moderate stimulation than it is to revive it if it has commenced to fail decidedly. The dosage of brandy or whiskey in typhoid fever at the age of 2 years may vary from $\frac{1}{2}$ to 1 fl. dram (2 to 4) every 3 to 4 hours, depending upon the urgency. Cardiac weakness may call also for digitalis, camphor, caffeine or similar drugs. This is true, however, only of the bad cases.

Vomiting, if severe, requires that the milk be alkalized, diluted, skimmed, peptonized, or entirely replaced for a time by other food, such as albumen water, cereals, or broths free from fat. Frequently it renders it advisable to give drugs hypodermically as far as possible. *Diarrhea* need not be interfered with if there are only 4 to 5 moderate-sized stools daily. If more numerous, or if large and watery, the condition can generally be controlled by small doses of silver, bismuth, salol, some of the tannic acid derivatives, or opium. The last-mentioned drug must be given cautiously if there is any tendency to coma or to decided tympany. *Constipation*, although often requiring treatment, is generally not troublesome. No purgatives should be administered except early in the attack. Small glycerine or larger soap-and-water enemata may be employed. Glycerine suppositories are often useful. It need scarcely be said that any injections must be given gently without undue pressure.

Tympanites is occasionally sufficiently annoying to demand relief. The local application of turpentine stupes is generally sufficient to relieve it. In other cases the careful insertion of a rectal tube is of benefit. Sometimes injections of soap-and-water or of milk of asafetida are useful. Asafetida by the mouth is frequently serviceable in this condition.

Nervous symptoms at times require treatment. As already stated, hydrotherapy is to be employed not so much for the mere reduction of temperature as for the alleviation of the attendant nervous phenomena. Delirium, sopor, great restlessness, headache, and the like, if associated

with high temperature, are often benefited by hydrotherapy. In other cases we may use small repeated doses of bromides or of antipyrine or phenacetin. Sleeplessness may well be combated by bromides or veronal. A warm bath in the evening may suffice to relieve it.

Complications and sequels need treatment appropriate for them. In intestinal hemorrhage the foot of the bed should be elevated, an ice-bag applied to the abdomen, and morphine given hypodermically in sufficient dose to quiet intestinal peristalsis. Epinephrine in doses of 5 to 10 minims (0.31 to 0.62) of the 1:1000 solution, given by the mouth or subcutaneously, calcium chloride or lactate (5 to 10 grains (0.324 to 0.648) 4 times daily) or gelatine (10 per cent. solution) may be tried. Intestinal perforation demands operative interference at the earliest possible moment. The debility and anemia which often persist after severe cases of typhoid fever require medication with iron, strychnine and other tonics, and often a sojourn at the seashore or in the mountains.

PARATYPHOID FEVER

This is a condition which has come into considerable prominence in recent years, and to which brief reference must be made. The disease is produced by the action of the paratyphoid bacillus, either of the varieties "A" or "B" being the agent. The latter is that most frequently found. Symptomatically the disorder almost exactly resembles typhoid fever, the chief distinction being that the agglutinative reaction with the typhoid bacillus is absent, while it is obtained with the variety of the paratyphoid bacillus which is the causative factor in the case. The affection may occur isolated or in small epidemics, and may affect any age. It appears to be uncommon in the 1st year of life, but an instance of the A type occurring in an infant of 8 months is reported by Eckert¹ and a number of instances by others of infants with Type B. A congenital infection by the B bacillus was observed by Nauwerck and Flinzer.² The lesions appear to be very similar to those of typhoid fever. Decided ulceration of Peyer's patches is uncommon, but this is equally true of typhoid fever in early life. Moreover hemorrhage and perforation have been reported. As far as experience has yet extended the mortality appears to be decidedly less than that of typhoid fever. The disease as it occurs in infancy and childhood has been exhaustively reviewed by Cannata.³

CHAPTER X

CEREBROSPINAL FEVER

(Epidemic Cerebrospinal Meningitis)

Although localizing itself largely upon the cerebrospinal meninges, and belonging with other forms of meningitis, the disease is so manifestly infectious and often epidemic, with a complex of symptoms so peculiarly its own that it seems properly included with others in the category of Infectious Diseases. It probably existed at a much earlier period, but it was first clearly described by Vieussieux in 1805⁴ in Geneva, and

¹ Berl. klin. Woch., 1910, XLVII, 1102.

² Münch. med. Woch., 1908, LV, 1217.

³ Annali di Clinica Medica, 1911, II, 285.

⁴ Hufelands, Journ. d. pract. Arzneymunde, 1805, XIV, 3 St., 181.

shortly afterward in the United States (Danielson and Mann;¹ Strong²); and since then has appeared in different countries with varying frequency.

Etiology. Predisposing Causes.—Climate and season exert a decided influence, the disease being confined to temperate climates and the majority of epidemics beginning in cold weather. Defective sanitation in general is likewise important, and outbreaks are consequently peculiarly liable to occur among soldiers in camps. Trauma of the head, exposure to heat, and mental and physical over-exertion certainly predispose. The previous health and the existence of other diseases are without direct influence.

Age is a powerful etiological factor, children and adolescents being especially susceptible, and infants in the 1st year being in no way exempt. Of 2915 cases in the Silesian epidemic in 1905 reported by Flatten³ 8 per cent. occurred in the 1st year, 47 per cent. from birth to 5 years, and 29 per cent. from 5 to 10 years. Of 2179 cases in the epidemic in New York City in 1904 and 1905 (Billings)⁴ 15 per cent. were under 1 year and 67 per cent. under 10 years of age. It has been observed even in the new born (Commandeur and Nordmann).⁵ Of the *individual susceptibility* little can be said with certainty. It is undoubtedly slight, since comparatively so few of those exposed are attacked. As a rule but a single case occurs in a family, although 2 or 3 or even more cases in a house are sometimes seen. *Epidemic influence* is very marked. Years may pass with but few cases in a locality, and then an outbreak may occur. These epidemics are generally limited in extent, perhaps to one city, while at other times a considerable part of a country, or even several countries, may be involved. The outbreak may continue for months or years and then cease entirely, or only sporadic cases develop. The severity of epidemics varies greatly in different localities and on different occasions.

Exciting Cause.—The disease is clearly an infectious one, now believed to be due to the *diplococcus intracellularis meningitidis* described by Weichselbaum⁶ in 1887, for although a symptom-complex resembling that of cerebrospinal fever may undoubtedly be produced by other germs, the disease occurring as a primary affection is generally dependent upon the meningococcus (Councilman),⁷ and cases with other bacteriological relationships are better classified under Simple Acute Meningitis. (See Vol. II, p. 320.)

The germ was first obtained by lumbar puncture from patients during life by Heubner⁸ who succeeded also in producing the disease in animals by inoculation. It is a diplococcus, different in form from the pneumococcus, and having many resemblances to the gonococcus, failing, as this does, to stain by Gram's method, although not so invariably. It is found in large or small numbers in the inflammatory exudate, chiefly within the cells; frequently on the nasal and pharyngeal mucous membrane, but only, as a rule, comparatively early in the attack; and sometimes in the blood. Goodwin and v. Sholly⁹ found it in the nose in the first 2 weeks in 50 per cent. of the patients. It has been discovered

¹ Med. & Agricult. Registry, Bost., 1806. Ref., Osler, Pract. of Med., 1903, 101.

² Dissert on the Disease Termed Spotted Fever, 1810.

³ Klin. Jahrb., 1905-6, XV, 211.

⁴ Journ. Amer. Med. Assoc., 1906, XLVI, June 2.

⁵ Lyon med., 1907, CVIII, 1081.

⁶ Fortsch. d. méd., 1887, V, 573.

⁷ Journ. Amer. Med. Assoc., 1905, XLIV, 997.

⁸ Jahrb. f. Kinderh., 1896, XLIII, 1.

⁹ Research Lab. Dept. of Health, New York City, 1905, I, 177.

also on the conjunctiva. Of very important bearing upon treatment is the fact as pointed out by Gordon¹ and others that there are different strains of the meningococcus, as shown by their immunological reactions. Two types prevail, one responsible for 75 to 80 per cent. of the cases (Flexner).²

Vitality of the Germ.—Nothing is definitely determined regarding the *life history* of the germ outside the body. Its vitality appears to be slight. It grows badly on most culture media, and it is readily killed by low or high temperature, or by drying. Even from the spinal fluid the germ disappears soon; often long before the patient has recovered. Occasionally, however, it persists for months. In chronic cases of the intermittent form the germ may sometimes be found only during the exacerbations. Sometimes it is not discovered at all until the disease is well advanced.

Mode of Transmission.—The method of transmission of the disease is not definitely known. Soil and water appear not to be factors. Whether it is carried by the air to any extent is doubtful. Spread of the infection by domestic animals is possible. Only in rare instances has the conveyance by clothing been proven. Direct transmission from the sick to the well is the exception. I have never seen a case develop in a hospital ward in which other cases of the disease were under treatment. Spreading by the schools does not seem to occur.

It is a noteworthy fact, however, that the microorganisms have repeatedly been found on the respiratory mucous membrane of healthy individuals during the existence of epidemics, and it is very possible that dissemination occurs in this way. Goodwin and v. Sholly³ found them in the nose in 10 per cent. of those in contact with patients, and Kutscher⁴ in the nasal and pharyngeal mucus in 75 per cent. Other investigators think that infection by the food is probable. The *mode of entrance* of the germ into the body is also unknown. The fact that it is often found on the nasal mucous membrane of patients has led to the belief that it reaches the meninges directly, by penetrating the cribriform plate of the ethmoid bone; others believe its entrance is by the way of the lymph vessels from the nasopharynx; and still others, that the portal of entrance is the intestine.

Pathological Anatomy.—The characteristic lesion is an acute fibrino-purulent inflammation of the pia-arachnoid of the brain and spinal cord. In malignant cases, fatal within a few hours, the dura and pia appear merely intensely congested, swollen, and possibly cloudy. Cellular infiltration may perhaps be discoverable only with the microscope. In cases lasting 2 to 3 days only a small amount of purulent exudate is found. In the severe cases which have continued a longer time subarachnoid exudate is evident; at first simply cloudy, but later usually distinctly fibrino-purulent. Over the cortex it occurs oftenest in streaks following the fissures and vessels; or it may be in the form of scattered yellowish or greenish-yellow plaques, sometimes covering the greater part of the convexity. It is generally most abundant at the base, forming a uniform yellowish layer with much thickening of the meninges. The choroid plexus is involved, and the ventricles are dilated by cloudy or distinctly purulent fluid. On the cord it is situated chiefly

¹ Kennedy and Worster-Drought, Brit. Med. Journ., 1917, II, 261.

² Jour. Am. Med. Assoc., 1918, LXXI, 638.

³ Journ. Infect. Dis., 1906, Suppl. Vol., 21.

⁴ Med. Klin., 1907, III, 314.

over the posterior portion and especially in the regions below the cervical. The spinal nerve-roots and the sheath of the cranial nerves may be surrounded by the exudate.

The exudate consists chiefly of polymorphonuclear leucocytes in a fluid, which, although more or less fibrinous, is never so to the extent seen in pneumococcal meningitis. In the acute cases large cells are also discovered, probably derived from the connective tissue or the lining of the lymph spaces. Meningococci, mostly within the cells, are present in the exudate as well as in the edematous meningeal tissue which is found between the areas of distinct cellular infiltration. In chronic cases, running a course of a month or more, the exudate has disappeared to a large extent, and its purulent character given place to a condition of a more mucous appearance. The meninges are left edematous and thickened and the ventricles may be greatly dilated.

The brain-tissue itself is affected to some extent, being congested and softer than normal, and exhibiting cellular infiltration together with meningococci in the superficial layers and especially along the vessels. In chronic cases cocci are scarce and found only with difficulty. The cranial nerves are infiltrated. The spinal cord shows similar changes but with fewer cocci. The nerve-roots and nerve-ganglia also exhibit evidences of inflammation.

The bones of the skull and the dura mater of the brain and cord are intensely congested. The spleen may be enlarged but is less often so than in most other acute infectious diseases. The lungs may exhibit bronchitis, hypostatic congestion, or the lesions of a complicating pneumonia, the consolidation being in the form of quite small foci consisting of purulent infiltration sometimes distinctly hemorrhagic (Councilman, Mallory and Wright)¹ and not developing in connection with the bronchi. These foci may be scattered or massed in the form of croupous pneumonia.

The lesions of endocarditis or pericarditis may occasionally be found. The heart, liver and kidneys exhibit degenerative changes, and sometimes the lesions of nephritis are present. Ecchymoses in the skin, punctate hemorrhages in the endocardium, abscesses in various parts of the body including the joints, suppuration in the internal ear, and inflammation of the eyeball are sometimes met with.

Symptoms. ORDINARY FORM.—The clinical picture of the disease varies greatly in different subjects, but certain clearly defined forms are recognizable. A general description of the ordinary type follows:—The duration of *incubation* is not definitely known. In a few cases Netter² reported it as between 3 and 11 days, and Flatten,³ in his studies on the Silesian epidemic, from 3 to 4 days or less. Bolduan⁴ placed it at from 1 to 4 days. It seems impossible to reach a positive conclusion. The attack may be ushered in by *prodromes*, lasting 1 or 2 days, and consisting of malaise, headache, vertigo, chilliness, pain in the back, and loss of appetite. As a rule, however, the *onset* is sudden, with fever, severe headache, prostration, vomiting, severe pain in the neck, back and limbs, and sometimes convulsions (Fig. 110). Very rapidly a peculiar degree of stiffness of the neck develops, and in well-marked cases decided retraction of the head as well. The slightest forcible moving of the head causes a cry of pain. Delirium, which is sometimes violent, great rest-

¹ Epidem. Cerebro-Spin. Meningitis, 1898.

² 20th Cent. Pract. of Med., XVI.

³ Klin. Jahrb., 1905-6; XV, 211.

⁴ Research Lab., Dept. of Health, N. Y. City, 1905, I, 140.

lessness, and irritability are common. Sensitiveness to light and noise, and cutaneous hyperesthesia are marked. As the disease advances vomiting, pain, and irregular fever continue, and more or less opisthotonos develops. There is often rigidity of the extremities. The face is congested, strabismus or alteration of the pupils appears, sleep is disturbed, and grinding of the teeth or general convulsions may occur repeatedly. There is frequent crying out with pain. Herpes or other eruptions of the skin may appear, if they have not done so at the beginning, prominent among them being those of a purpuric nature. As intracranial pressure increases the pulse becomes slow and often irregular, the respiration irregular, stupor replaces delirium, and complete coma follows.

The *duration* of the disease, even in what may be called average cases, is extremely variable. Roughly speaking it may be placed at from 2 to 4 weeks, not including convalescence, but may be much shorter or longer than this. Improvement is characterized by the diminution of the pain and rigidity, permanent lessening of fever, cessation of vomiting and improvement of the mental state.

Some of the symptoms must be considered more in detail.

Convulsive movements are frequent in infancy and early childhood; less so in older persons. They may be general, and usher in the attack and then cease; or recur at intervals later, being then either general or local. Sometimes convulsions occur from time to time in the chronic cases after most other symptoms have disappeared. Grinding of the teeth is common and tremor may occur.

Pain is a very common symptom, beginning early and continuing throughout the acute portion of the attack. It is situated chiefly in the head, but may involve also the back, abdomen and the limbs, especially the lower. It is subject to sudden exacerbations, especially at night, and is often so distressing that it occasions loud outcries (the "hydrencephalic cry"). It is especially marked on any forcible movement of the body. *General hyperesthesia* is very constant, the patient being greatly disturbed and often crying out on hearing a loud noise, being exposed to bright light, or on the mere touching of the skin.

Muscular rigidity is almost always seen, chiefly in the form of stiffness of the neck and some degree of retraction of the head (Fig. 111). If the head is lifted forcibly from the pillow the trunk follows it without any bending of the neck taking place. It can, however, be turned from side to side without difficulty. In severe, long-continued cases the occiput may even press against the back beneath the scapulæ (Fig. 112). It not infrequently happens, however, that the stiffness of the neck is intermittent, not being discovered at one examination although present at another. Rigidity with anterior curving of the spine is common, and the children often lie on the side with the arms flexed stiffly and drawn over the chest, the legs flexed, and the thighs drawn to the abdomen;—the so-called "gun-hammer" position. The muscles of the face may be tense and the *risus sardonicus* present (Fig. 113). Trismus may occur, the abdomen is often scaphoid, and Kernig's sign—viz., the inability to extend the leg by passive movement when the thigh is at right angles with the trunk—is generally observed, as in all forms of meningitis. The *tendon reflexes* are uncharacteristic, being either normal, increased or absent.

The *mental symptoms* are variable. Great restlessness is common early in the attack and may persist. The mind may be clear much of the time, but delirium is common and may be intense or even maniacal,



FIG. 111.—OPISTHOTONUS IN CEREBROSPINAL FEVER.

Boy of 3 years in the Children's Hospital of Philadelphia. See history with Fig. 117.



FIG. 112.—OPISTHOTONUS IN THE SUBACUTE STAGE OF CEREBROSPINAL FEVER.

Boy of 1 year, ill for 4 or 5 weeks with irregular temperature, emaciation, leucocytes 33,500, spinal fluid under great pressure and almost clear, 300 cells to the c.mm., 80 per cent. polymorphonuclears, meningococci, death.



FIG. 113.—RISUS SARDONICUS IN CEREBROSPINAL FEVER.

Infant of 7 months in the Children's Hospital of Philadelphia. Died after 15 days of illness. Photograph taken on 21st day. Shows the facies as well as the spastic condition of the extremities.

the child tossing wildly about the bed, or even jumping out of it. In other cases it is merely of the wandering type, and either constant or intermittent; or it may be followed or replaced even at the onset by a more or less apathetic or even stuporous condition. The degree of delirium does not appear always to bear any definite relationship to the other symptoms or to the gravity of the attack in general. In severe cases coma is liable finally to supervene, or it can even be one of the earliest symptoms. As in the case of delirium, it may vary greatly from day to day, sometimes rapidly disappearing or reappearing; or coma and delirium may alternate. The expression of the face in the acute condition is that of excitement and irritability, except in the mild cases.

Among *digestive disturbances* vomiting is an early symptom, present in the majority of cases. It may be frequent enough to debilitate the patient greatly. Generally it subsides as the disease advances, but to



FIG. 114.—PURPURIC ERUPTION IN CEREBROSPINAL FEVER.

Boy of 6½ years, a patient in the Children's Ward of the University Hospital, Philadelphia. Rash appeared on the 4th day of the disease, abundant on all extremities. Case a severe one; improved temporarily, but terminated fatally.

this there are many exceptions. It is cerebral in origin, and may or may not be attended by coating of the tongue. Appetite is diminished. Constipation is generally present.

The *temperature* is irregular and entirely uncharacteristic. It generally rises rapidly, and it then remains high or diminishes; but sudden remissions or intermissions as well as sudden rises to 105°F. (40.6°C.) or over are liable to occur. A temperature of 101 to 103°F. (38.3 to 39.4°C.) is an average one. Some patients never exhibit much fever. In fatal cases unusual hyperpyrexia is sometimes seen (Fig. 117). As a rule, however, there is little connection in acute attacks between the height of the fever and the severity of the disease. Irregularity is especially marked as convalescence goes on, or as the disease passes into a chronic state.

The *pulse* bears little relationship to the temperature. It is usually more rapid than normal, especially if there is great general debility. It is subject to sudden changes in rate, and may be slow or irregular if intracranial pressure is increasing. The arterial tension is low.

The *respiration* is not characteristically affected. Its rate may be decidedly increased by the presence of pain. In advancing cases it may become sighing, irregular, or even approaching the Cheyne-Stokes type.

The *cutaneous symptoms* are interesting; the most frequent generally

being herpes, which is present in a large proportion of cases. It is usually situated on the face, but sometimes elsewhere. As a rule an early symptom, it may not develop until later, or it may come out in crops. A petechial or a purpuric eruption is common, its frequency varying greatly with the epidemic (Fig. 114). In the New York outbreak of 1905 it was recorded in 19 per cent. of the cases; while in the earlier epidemics in the United States it was so common that it gave rise to the title of "Spotted

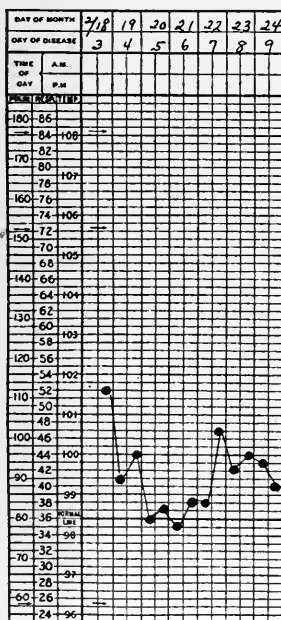


FIG. 115.

FIG. 115.—CEREBROSPINAL FEVER, ABORTIVE FORM.

Milton L., 2 years old. Onset with repeated convulsions, prolonged unconsciousness, delirium, fever. 2d day showed stuporous condition and fever, the child being apparently very ill. Rapid improvement followed, the mind becoming quite clear by the 4th day, and convalescence being entirely established by the 8th or 9th day. Meningococci found in the spinal fluid.

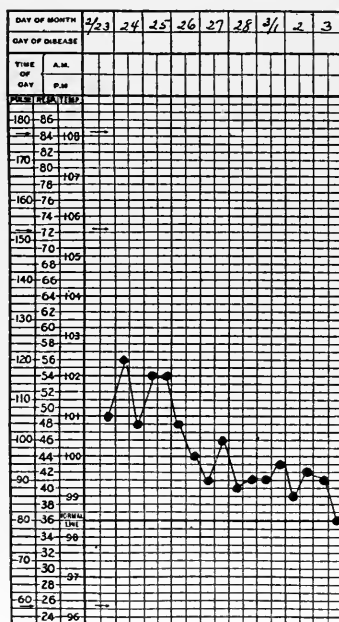


FIG. 116.

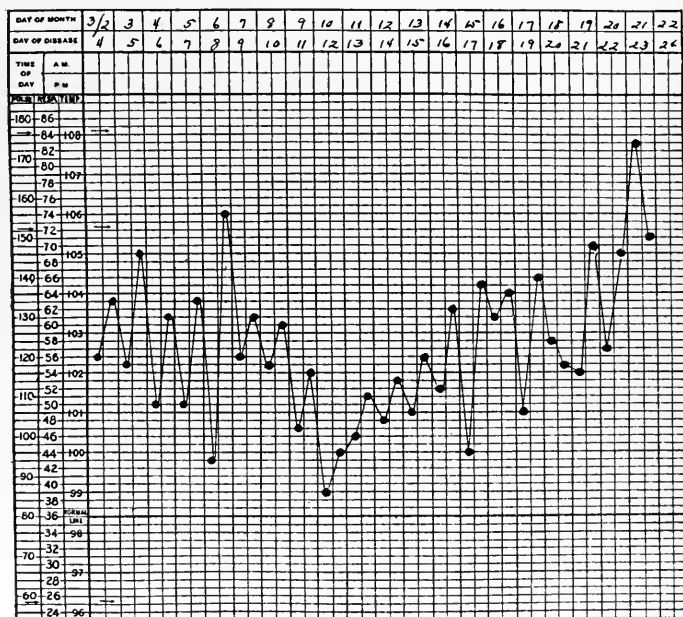
FIG. 116.—CEREBROSPINAL FEVER, MILD FORM.

Mary K., aged 5 years. One of three children of the family ill with the disease. Exact date of onset uncertain, but had been slightly ill for not over a week. Feb. 23, sleeps much of time, mind seems entirely clear, expression placid, apparently no pain, no irritability, no hyperesthesia, abdominal tache marked, neck slightly stiff, head slightly retracted but only if the child lies on her side, no other rigidity, no herpes or petechiae; Feb. 26, improving, much brighter; Feb. 28, greatly better, stiffness of neck gone; Mar. 10, out of bed, entirely well. No serum used. Children's Hospital of Philadelphia.

Fever." In the epidemic in Philadelphia in 1917 and 1918 I observed it in comparatively few cases. It may appear early or later, and seems to bear little relation to the severity of the attack. In some cases larger cutaneous hemorrhages occur. A well-marked *tâche cérébrale* is a common symptom, as in all forms of meningitis, and often there may be noted an irregular flushing of the trunk when exposed, or of the face, the evidence of the vasomotor disturbance present (see Vol. II, p. 322, Fig. 340).

In any case at all long-continued *emaciation* is very decided and in chronic cases is liable to become extreme.

The *blood* always presents an early and very decided leucocytosis, especially of the polymorphonuclear cells, equalling sometimes as much as 40,000 or more to the c.mm. The eosinophiles disappear. The *urine* is normal or exhibits a febrile albuminuria. Small amounts of sugar are occasionally observed.



VARIATIONS FROM THE ORDINARY FORM.—Even among cases of the ordinary type there is the greatest variation in the symptoms. Some begin with very severe manifestations which soon ameliorate. The patients, however, may fail to convalesce at once, but pass perhaps through weeks of illness of much diminished severity. Other cases begin mildly but soon grow more severe. Others reach a fatal ending through some one of the numerous complications which are prone to develop. The variations are often so decided that a number of special types are described. Among these may be mentioned (1) the Abortive form; (2) the Mild form; (3) the Severe form; (4) the Malignant form; (5) the Chronic form; and (6) the Intermittent form.

1. Abortive Form.—In this variety the disease begins abruptly and severely, but in 2 or 3 days the threatening symptoms disappear and the patient rapidly recovers (Fig. 115).

2. Mild Form.—In cases of this sort the symptoms are mild from the outset, or soon become so after a severe onset (Fig. 116). There may be only slight headache and nausea, occasional vomiting, slight stiffness and pain in the neck, and little fever. The mind is clear, or nearly so. The patient may not even be confined to bed. Sometimes the symptoms are so trivial and uncharacteristic throughout that diagnosis would be impossible if the case were an isolated one. In 1 case of 3 occurring simultaneously in a family under my care the only symptoms present were slight fever and cerebral *tâche* and a slight rigidity of the neck; all of which would have passed unnoticed had the case occurred alone. In another instance, also 1 of a family group of 3, the patient was suffering from a mild attack of pneumonia; only the *tâche* and the very moderate rigidity of the neck indicating that this disease was a complication of a very mild cerebrospinal fever.

3. Severe Form (Fig. 117).—In this all or many of the symptoms are intensified. The type does not differ materially from that described as the ordinary variety, except for a greater severity of the manifestations. The course may be short or prolonged.

4. Malignant or Fulminating Form (Figs. 118 and 119).—This variety is characterized by the extremely sudden onset, the intensity¹ of the symptoms, the tendency to severe collapse, and the shortness of the course. The child may be stricken suddenly while at play. It may suffer from repeated convulsions and die in less than 24 hours, the diagnosis being impossible unless the case be one of a family group or a lumbar puncture be made. Sometimes coma and collapse are the earliest or the only symptoms, or there may be most violent repeated vomiting or intense headache. Widespread cutaneous hemorrhages may develop and hemorrhage from the mucous membranes take place.

5. Chronic Form.—This might be called one of the terminations of the disease. The symptoms at first do not differ from those of the ordinary type. Instead, however, of disappearing gradually, they continue in a modified form. Fever may be absent for a considerable time and all the symptoms may ameliorate. Then, with the recurrence of elevated temperature the stiffness of the neck increases, vomiting returns, delirium or stupor reappears, and convulsions may occur. This condition may last a variable time, to be followed apparently by the beginning of certain convalescence, when a second recurrence takes place. In this way the disease may be protracted for months. Not all the symptoms mentioned need be present. In fact, the symptomatology is likely to be very variable. In some cases the remissions in tempera-

nent disability. The most important are those affecting the nervous system and the special senses. The *eyes* may exhibit neuritis of the optic nerve, due to involvement by the exudate at the base of the brain, or the purulent process may extend along the pia-arachnoid of the nerve and produce a purulent choroido-iritis. In other cases a neuritis of the fifth nerve is followed by purulent conjunctivitis or keratitis. These

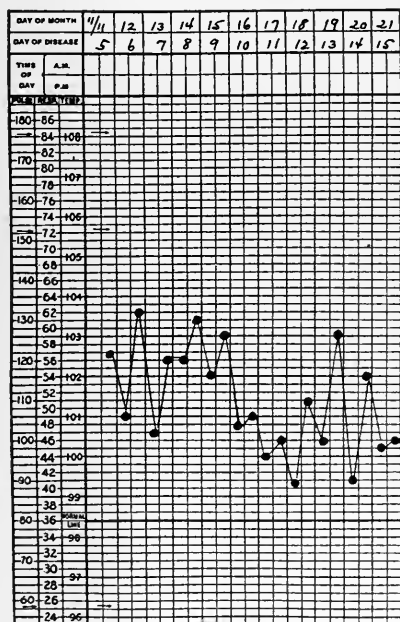


FIG. 120.

FIG. 120.—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R., 6½ years old. Children's Hospital. Chart of 5th to 15th day inclusive.

I. *Acute Stage*.—Nov. 11, 1904. Irritability, delirium, pain in neck, back and limbs, strabismus, rigidity, retraction of head, hyperesthesia, leucocytosis 19,520. Lumbar puncture showed thick pus with meningococci; Nov. 16, gradual improvement; Nov. 18, rational to a considerable degree; Nov. 21, petechiæ; Nov. 26, improving decidedly, slight hyperesthesia and some rigidity and pain remaining.

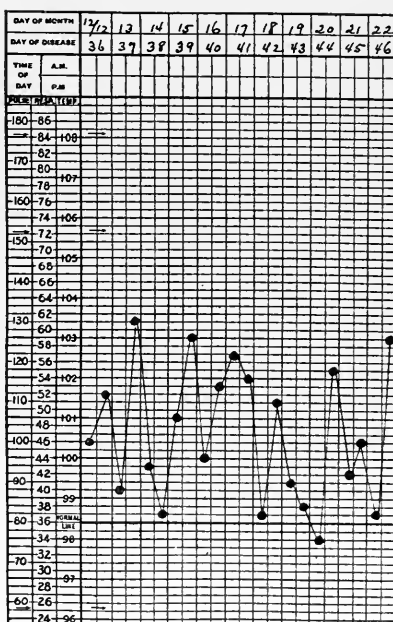


FIG. 121.

FIG. 121.—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (Continued). Chart of 36th to 46th day inclusive.

II. *Partially Intermittent Temperature*.—General condition in December much improved. Still strabismus, slight stiffness of neck, and frequent pain in the limbs and back. Began to walk by the end of December.

lesions may develop early or later in the attack. Complete or partial blindness may result. The *ears* are very often involved. Westenhöffer¹ believes that otitis media is present in all cases in children. If purulent it may be the cause of loss of hearing. Absolute deafness from inflammation of the labyrinth, resulting from extension of the process along the auditory nerve, is a not infrequent sequel. Moos² found 38 deaf-

¹ Klin. Jahrb., 1905-6, XV, 657.

² Die Taubstummheit in ihrer Abhängigkeit m. Cerebr.-sp. Mening., 1883. Ref., Councilman, Mallory and Wright, *loc. cit.*

muters in 64 recovered cases of cerebrospinal fever. Probably the majority of cases of deaf-mutism in institutions owe their origin to this disease.

Disordered mental states may occur as sequels, among them being aphasia and mental impairment. Among 539 cases of mental defect in Norway, reported by Looft¹ 3.7 per cent. resulted from cerebrospinal fever. *Headache* is sometimes very persistent. *Hydrocephalus* is a serious and very frequent sequel. Undoubtedly many of the chronic cases of cerebrospinal fever are due to it. The group of symptoms characterizing it

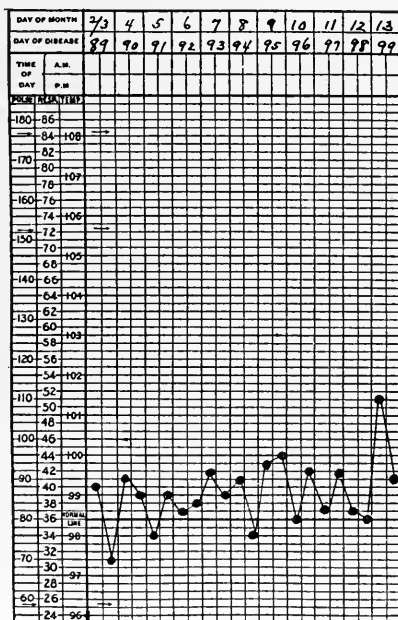


FIG. 122.—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (Continued). Chart of 89th to 99th day inclusive.

III. *Apparent Convalescence, Followed by Some Return of Symptoms.*—During last of December and January child constantly improving. Out of bed and walking about the room. Strabismus and slight rigidity of neck remained. Last of January frequent vomiting began. Occasionally slight convulsions. Child looked less well. Leucocytes increased from 14,350 on Feb. 1 to 17,050 on Feb. 13. Practically no fever.

as outlined by Ziemssen² and others, consists of vomiting; severe pain in the head, neck, and limbs; rigidity; great emaciation; increasing apathy; convulsions; and finally coma. This condition may alternate with periods of decided improvement in health. Hydrocephalus of an acute form may occur even early in the disease. This would account for some of the symptoms observed at that period. *Paralysis* may involve the eyes or the face; less often the limbs, in the latter case being either hemiplegic or paraplegic in type. It may be temporary or permanent. It generally does not develop until well on in the attack.

Inflammation of the pharynx, nasopharynx, and tonsils is a frequent

¹ Nord. med. Ark., 1901, II, No. 4.

² Hand. spec. Path. u. Therap., Bd. II, Th. II, 683.

complication occurring early in the attack, and sometimes antedating other symptoms. *Pneumonia* is frequently combined with meningitis. When it is the primary disease it is pneumococcic in origin and the meningitis is probably of the same nature and not to be classed as cerebrospinal fever. *Pneumonia*, however, is frequently seen as a complication secondary to cerebrospinal fever, and probably is produced by the meningococcus. *Nephritis* is a serious complication occasionally seen adding to the gravity of the case. Although not reported so frequent

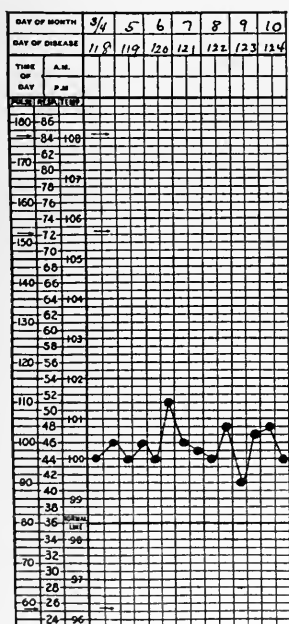


FIG. 123.

FIG. 123—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (Continued). Chart of 118th to 124th day inclusive.

IV. *Continuance of Subacute Symptoms*.—Mar. 1, child again confined to bed. Frequent distressing pain in head, neck, and limbs, emaciation decided, losing power in limbs, irritable and capricious but mind clear, slight febrile reaction. Sedatives constantly required.

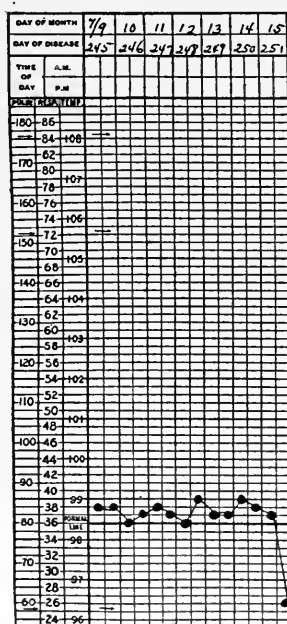


FIG. 124.

FIG. 124—CEREBROSPINAL FEVER, CHRONIC FORM.

Willie R. (Continued). Chart of 245th to 251st day inclusive.

V. *Gradual Increase of Hydrocephalic Symptoms*.—Slow but constant loss of strength continued, pain very frequent and distressing, emaciation extreme. In July, failed rapidly, rigid all over, head much retracted, periods of unconsciousness and finally persistent coma. Died July 15, on the 251st day of the disease.

by most writers, Steiner and Ingraham¹ found evidence of its presence in 28 out of 145 cases of the disease. *Arthritis* affecting a number of joints is a not infrequent complication or sequel. The fluid may be serous or purulent in nature and contain the meningococcus. In other cases the joint is red and swollen without evidence of effusion.

Cerebrospinal fever may occasionally occur simultaneously with or as a sequel to *other acute infectious diseases*, among these being typhoid

¹ Amer. Journ. Med. Sci., 1908, CXXV, 351.

fever, scarlet fever, measles and diphtheria. Collins¹ reports an instance of the combination of this disease with malaria.

Among other complications sometimes seen are pleurisy, pericarditis, endocarditis, peritonitis, parotitis, enteritis, subcutaneous abscesses, urticaria, pemphigus, and erythema.

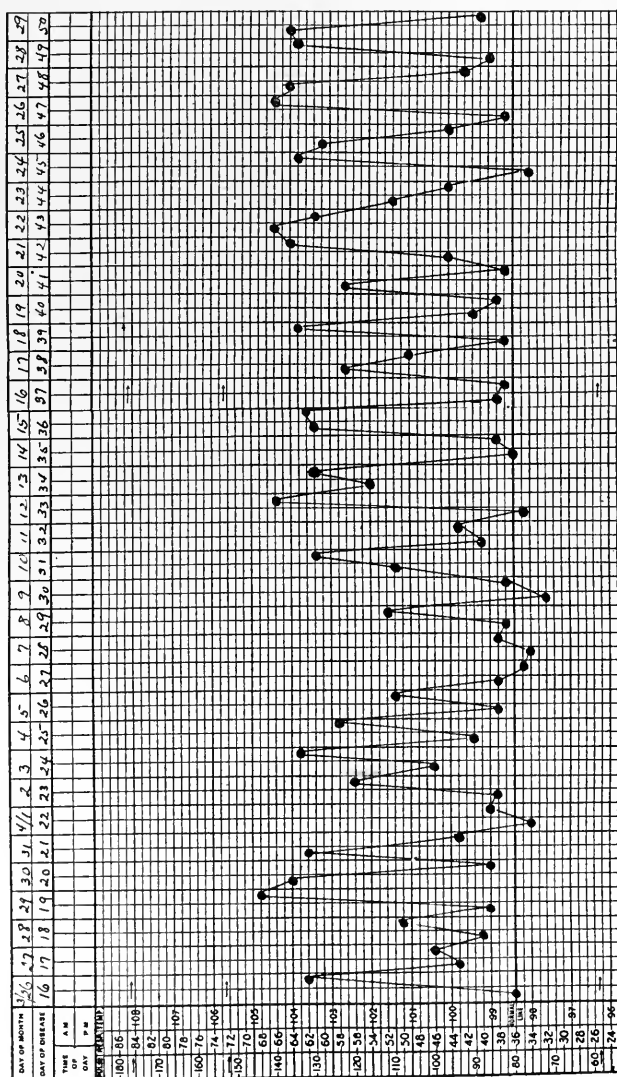


FIG. 125.—CEREBROSPINAL FEVER, INTERMITTENT FORM.

Regina J., aged 4 years. Portion of chart illustrating approach to the intermittent type of the disease. The rises of temperature were generally attended by renewal of projectile vomiting, and often by pain in the neck and elsewhere. Recovery.

Relapse.—In a disease with such an uncertain course and with such a natural tendency to recrudescence after convalescence is apparently beginning, it is difficult to reach any conclusion regarding the frequency of relapse. Indeed, the disposition to it may be called a part of the disease.

¹ Boston Med. and Surg. Journ., 1911, CLXV, 610.

Recurrence.—Little is known about recurrence. As a rule one attack appears to protect from subsequent ones, although instances to the contrary are reported.

Prognosis.—The disease is a serious one, the mortality varying according to Hirsch¹ from 20 per cent. to 75 per cent., and being oftener nearer the latter figure than the former. The actual number of cases dying during epidemics is often large, over 2500 having succumbed in New York City in the outbreak of 1904-1905 (Billings).² The mortality is especially high in early life. Friis³ found it in the 1st year to be 77.7 per cent., from 1 to 5 years, 48.7 per cent., from 5 to 10 years 51.6 per cent., and from 10 to 15 years 21.4 per cent. Of 779 fatal cases collected by Hirsch⁴ 208 were under 1 year of age, 337 from 1 to 5 years, 151 from 5 to 10 years, 41 from 10 to 15 years, 16 from 15 to 20 years, and 26 over 20 years of age. It is impossible to predict what the outcome will be in any given case, and the prognosis must always be very guarded. The fulminant cases are nearly always fatal. Sudden, severe onset; very exhausting and continued vomiting; persistent, unusually high temperature; the repeated occurrence of convulsions; rapid pulse and respiration, and the early development of coma or its long persistence are unfavorable symptoms. A drop in the number of neutrophils in the blood and an increase of the lymphocytes is a favorable indication. Death takes place oftenest within the 1st week, and the outlook is consequently brighter when the 2d week is well under way and the symptoms seem to be ameliorating. To this, however, there are numberless exceptions, since a long-continued chronic form frequently develops which ends fatally oftener than in recovery. There is always, too, the very great danger of permanent sequels remaining. Epidemic influence upon the mortality is marked, the number of deaths being much greater in some years than in others. More fatal cases occur at the beginning of an epidemic than later.

What has been said above regarding the mortality-rate applies only to the condition before serum-treatment was commenced. This will be discussed under Treatment.

Diagnosis.—This rests especially on the sudden, severe onset, vomiting, delirium, restlessness, intense headache, stiffness and pain in the neck, retraction of the head, pain in and rigidity of the muscles of the back and limbs, leucocytosis, herpes, the rapid development of the symptoms early in the attack, and the great tendency to variation in its course. All these are indications of the presence of some variety of meningitis, but in no other is there such a striking complex of symptoms. The spinal manifestations are generally more marked than in any other form. In the malignant cases the diagnosis may be impossible. This is true also of the very mild cases without suggestive symptoms. The existence of an epidemic is often of great service in reaching a conclusion, but the individual character of the epidemic is to be borne in mind. In that in Philadelphia in 1917 the diagnosis, although easy in many cases, was in others very confusing. In some instances the early symptoms were so entirely uncharacteristic that only the fact that the disease was prevailing led to the performing of lumbar puncture, and the discovery, based only on this, that meningitis existed. Percussion of the skull, recom-

¹ Die Meningitis cerebrospinalis epidemica, 1866, 33.

² Journ. Amer. Med. Assoc., 1906, XLVI, June 2.

³ Ugerskrift for Laeger, 1891, Ref. Netter, 20th Cent. Pract. of Med., XVI.

⁴ Loc. cit.

mended by Macewen, for the discovery of fluid in the ventricles is strongly urged by Koplik,¹ a slightly tympanitic note in the lateral regions indicating the presence of fluid.

The character of the spinal fluid is most important diagnostically. The early purulent nature distinguishes it from tuberculous meningitis, in which the fluid is clear or only slightly opalescent. There is the contrast, too, between the numerous polymorphonuclear cells of cerebrospinal fever and the lymphocytic cells usually predominating in cases of tuberculous meningitis. In the more chronic cases the fluid may be clear, but by this time the distinction from tuberculous meningitis can generally be made readily in other ways. The distinguishing of the fluid of meningococcic meningitis from that of other acute purulent forms can be done only by bacteriological study.

Cerebrospinal fever is to be differentiated from several other forms of meningeal inflammation. *Tuberculous meningitis* comes on, as a rule, very slowly and insiduously with only a later development of marked cerebral symptoms and finally of coma, and is without the rapidity of alteration in the symptoms to the degree characteristic of cerebrospinal fever. These diagnostic differences apply, however, only to typical cases of the disease; in others the differentiation based on symptomatology, apart from lumbar puncture, may be impossible.

Simple acute meningitis, in the sense of being due to germs other than the meningococcus, may simulate cerebrospinal fever so closely that diagnosis cannot be made except by lumbar puncture. Spinal symptoms, however, are generally less often present and the existence of some evident cause is often discoverable, since the disease is usually a secondary one. It is always, too, of a severe form and generally fatal; and consequently the diagnosis from milder cases of cerebrospinal fever is usually made easily. It seems very probable, as Still² has maintained, that the posterior basic meningitis first described by Gee and Barlow³ as occurring in infants is, in reality, cerebrospinal fever, since the microorganisms are practically identical.

Cerebrospinal fever may be confounded with several diseases which present no lesions of the meninges. Prominent among these is *typhoid fever* of the meningitic type. As a rule the passage of time will make the diagnosis easy, although there are certainly exceptions to this. The meningitic symptoms generally soon disappear in typhoid fever and other characteristics become evident, especially the absence of leucocytosis and the presence of the Widal reaction. The mere presence of leucocytosis, however, aids but little, since this may occur in typhoid fever as the result of some complicating condition. Occasionally *grippe* exhibits symptoms resembling those of cerebrospinal fever so closely that only lumbar puncture can settle the diagnosis. Yet, as a rule, cases of this disease with meningitic symptoms exhibit a shorter course and no retraction of the head. The high degree of leucocytosis in meningitis is also of diagnostic value in excluding *grippe*. It is to be borne in mind that a true influenzal meningitis is sometimes observed. *Pneumonia* may be ushered in with symptoms simulating meningitis. The examination of the blood does not aid, since both conditions exhibit a high leucocytosis. The course of the case will make the diagnosis clear. It is to be remembered, however, that an actual meningitis may occur in combination with pneumonia,

¹ Amer. Journ. Med. Sci., 1917, CXXXIII, 547.

² Journ. Path. and Bact., 1898, V, 147.

³ St. Barth. Hosp. Rep., 1878, XIV, 23.

either of a serous or less often a pneumococcic nature. In suspected cases lumbar puncture may settle the question as to the presence of meningitis and the character of any germs found. *Autointoxication* of gastrointestinal origin may begin with vomiting and meningeal symptoms and be the cause of considerable uncertainty of diagnosis for a time. As a rule however, the nature of the case soon becomes evident.

Treatment. Prophylaxis.—With our ignorance of the method of extension of the disease, preventive treatment seems as yet almost impossible. The isolation of affected persons is desirable in spite of the lack of evidence of direct communicability. The employment of mild disinfectant nasal sprays in the case of those known to have been exposed is also to be recommended, in view of the possible carrying of the disease by healthy persons.

Treatment of the Attack.—Except as regards the serum-treatment, this is largely symptomatic. The patient should be kept very quiet in a darkened room, in order to combat the excessive hyperesthesia. As the disease is a depressing one, the strength should be maintained by sufficient nourishment, not necessarily liquid, and often by alcoholic and other stimulants, given freely if need be. Although vomiting may render feeding difficult and a modification of the choice of food necessary, it is to be remembered that this symptom is cerebral in origin, not dependent upon indigestion. Comatose patients should be fed by gavage. Digitalis is frequently required. Strychnine is, in my opinion, better avoided, as it seems sometimes to increase the excitability. Bromides are often useful to quiet the patient, but the best drug for this purpose and for the relief of pain is morphine given hypodermically, taking care not to precipitate or increase a tendency to coma. Inunctions of mercurial ointment have been recommended. The employment of an ointment or of suppositories of colloidal silver has been advocated and may be tried. I have never been able to convince myself of any actual benefit being obtained.

Blisters to the back of the neck serve only to increase the discomfort. Warm baths at 100°F. (37.8°C.) are useful to quiet nervousness. An ice-bag may be applied to the head, remembering, however, the danger of depression by this treatment in the case of little children. In the later stages the administration of iodides is recommended to favor absorption of meningeal thickening.

The removal of the exudate by lumbar puncture often gives surprising relief if the symptoms indicate cerebral pressure. It should be employed in all such cases. The injection through the needle of a 1 per cent. solution of lysol has been recommended by Franca¹ but has not met with general acceptance. Inasmuch as the inflammation often shuts off the cranial cavity from the spinal cord, lumbar puncture frequently fails to relieve the cerebral symptoms. In such cases tapping of the ventricles was advocated by Schultz² and is often serviceable.

Serum-Treatment.—Kolle and Wassermann,³ Jochmann,⁴ Flexner and Jobling,⁵ Dopter⁶ and others have prepared a serum for use in this disease. That made by Flexner and Jobling is obtained from horses

¹ Deut. med. Wochenschr., 1906, XXXII, 609.

² Deut. Arch. f. klin. Med., 1907, LXXXIX, 547.

³ Deut. med. Wochenschr., 1906, XXXII, 609.

⁴ Deut. med. Wochenschr., 1906, XXXII, 788.

⁵ Journ. Exper. Med., 1908, X, 141.

⁶ Ann. de l'instit. Pasteur, 1910, XXIV, 96.

which have been repeatedly inoculated with cultures of meningococcus. After 20 c.c. (0.68 fl.oz.) or more of the exudate have been removed by lumbar puncture the serum, previously slightly warmed, is injected through the needle, which has been left in position. It may be given through a funnel and rubber tube, allowing it to enter by gravity, or a piston syringe may be used cautiously. The injection should always be made very slowly. The amount injected should always be less than that of the exudate removed, in order to avoid increasing the intracranial pressure. Elevation of the hips favors the flow of the serum to the

diseased region. The initial dose should be 10 to 15 c.c. (0.34 to 52 fl.oz.) for infants and not over 30 c.c. (1.014 fl.oz.) for older children. The injection should be made every 24 hours, sometimes oftener, during 3 or 4 days or more, beginning as early in the course of the attack as possible. The good results are sometimes immediate and surprising. The temperature in such cases may fall several degrees, the mental symptoms are greatly ameliorated, and the number of neutrophils in the spinal fluid is much reduced, and of the meningococci likewise. In most cases, however, even where there is reason to believe that the serum is exerting a beneficial action, there is, in my experience, no immediate decided effect upon the temperature discoverable. The treatment appears to produce also a diminished tendency to the development of sequels. When no fluid can be obtained by lumbar puncture, the serum, in the case of infants, may be injected into the lateral ventricles after withdrawing exudate from this region.

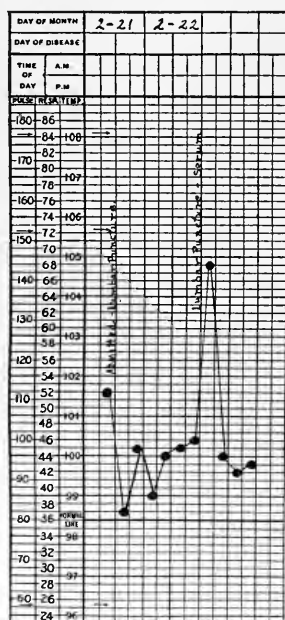


FIG. 126.—CEREBROSPINAL FEVER, WITH RISE OF TEMPERATURE AFTER INTRASPINAL INJECTION.

Frank F., 2 years old. Chart shows rise of temperature which sometimes occurs after serum-treatment. Children's Hospital.

increased restlessness, nervousness, and rise of temperature after injection (Fig. 126, and history, Fig. 117, p. 424). Consequently a careful continued observation of the state of the respiration and the general condition of the patient should be maintained during the procedure and after it. On the ground that the disease is in some cases primarily a sepsis the administration of the serum intravenously has been recommended, and has been done with good results (Herrick.¹)

As regards the general effect of the serum treatment upon prognosis, Flexner² found that whereas the usual mortality in various parts of the world had reached as high as 70 per cent., in 1294 cases receiving this treatment it had been reduced to 30.9 per cent. The period of the disease at which the treatment is commenced is also of influence. Of

¹ Jour. Amer. Med. Assoc., 1918, LXXI, 612.

² Journ. Exper. Med., 1913, XVII, 553.

Flexner's series it was begun in 199 in the first 3 days, with a resulting mortality of but 18.1 per cent. It is particularly in infancy that the good results are seen. Previously nearly all of this age died, especially subjects under 1 year old, whereas in 129 of Flexner's cases in the 1st year the mortality equalled 49.6 per cent. Between 5 and 10 years of age there were 218 cases with a mortality of 15.1 per cent. That serum-treatment is so efficacious in some instances and of so little value in others depends doubtless in part upon the severity of the infection in



FIG. 127.—SERUM RASH IN CEREBROSPINAL FEVER.

Child of 3 years in the Children's Ward of the University Hospital, Philadelphia. Urticarial eruption appeared on the 4th day after the first intraspinal injection of serum; lasted 4 days. Photograph shows widespread eruption over the face and body.

the individual case, and in part probably upon the strain of the meningococcus, to which reference has been made (p. 417). If the serum contains a strain which is not the one operative in the case, no benefit can, of course, be expected.

After the acute stage of the disease is over little benefit is to be expected from serum-treatment. Nevertheless it should by all means be tried, as occasionally good results follow. To avoid mistakes in diagnosis it must be remembered that the injection of serum is naturally capable of producing the same symptoms, including the cutaneous eruption, which occur from the injection of antidiphtheritic serum (Fig. 127).

CHAPTER XI

ERYSIPELAS

Although it could well be classified as a form of sepsis, erysipelas has such distinct peculiarities that it deserves separate consideration. At one time a greatly dreaded affection, especially in hospital practice, better sanitary methods have diminished its frequency very greatly.

Etiology.—The disease is widespread, climate and locality having no influence upon it. It appears to be more common in the cold season of the year and particularly in spring. Although often occurring in epidemics, and reappearing with especial frequency in certain localities, such as old hospitals not well cared for and in infant asylums, sporadic cases sometimes develop in institutions under the best hygienic regulations.

Age offers no protection, yet the disease appears to be more frequent at certain periods of life. In 1568 cases studied by Roger,¹ 27 occurred in the first 2 years of life; 6 at from 2 to 5 years inclusive; 60 at from 6 to 14 years; 189 at from 15 to 20 years, and 239 at from 21 to 25 years. In rare instances it has been acquired before birth. There is undoubtedly an individual susceptibility, some persons being particularly liable to it, and an inheritance of this susceptibility seems certainly possible. By far the most important predisposing factor is the presence of a *wound*. Consequently infants with an umbilical wound, eczematous areas, pustules of varicella, and the like, or those who have been recently circumcised or vaccinated, are especially predisposed. Sometimes the portal of entry is the mucous membrane, whence the disease spreads to the skin; yet erysipelas often develops without the slightest abrasion being discoverable anywhere. That there are not more individuals with wounds attacked indicates the lack of susceptibility in the majority of them.

Erysipelas is clearly an infectious disease, the germ being transmitted by direct contact or by clothing and the like, or by means of a third person. It is not diffused by the air to any extent. The *specific germ*, the streptococcus erysipielatis, first isolated and identified by Fehleisen² has been found to be a form of the streptococcus pyogenes. Its vitality is not great, since it lives in cultures not longer than 2 to 3 weeks. Occasionally other germs than the streptococcus appear able to produce the disease.

Pathological Anatomy.—The affected skin exhibits post-mortem an exudation of serum and of lymphoid cells in the cutis and subcutaneous tissue, with dilated and engorged blood-vessels, and with very numerous cocci in the lymph channels; these being most numerous in the region last involved. Sometimes large bullæ containing serum are found. In the most severe cases evidences of suppuration are observed in the subcutaneous connective tissue. Often alterations are present in other parts of the body, among these being enlargement of the spleen, parenchymatous changes in the liver and kidneys, infarcts in the lungs, kidneys and spleen, septic endopericarditis and pleuritis, lymphatic swelling, peritonitis, and occasionally meningitis.

¹ Arch. gén. de méd., 1901, CLXXXVIII, 5.

² Deutsch. Zeitschr. f. Chir., 1882, XVI, 391.

Symptoms. Incubation.—The period, of incubation is generally short, from 15 to 60 hours as proven by the inoculation experiments of Fehleisen.

Symptoms of the Attack.—Initial symptoms may be absent, or consist of chilliness, coldness of the extremities, restlessness, prostration, vomiting, high fever, and occasionally convulsions. Simultaneously, or nearly so, redness, swelling and tenderness of the skin develop in the affected area. Roger's statistics¹ show that the face is much most frequently the starting point. This was true of 83 per cent. of the cases at all ages. In 66 children of from 2 to 15 years the disease began in the lower extremities in but 4 instances. The redness exhibits the uniform flush of a dermatitis, which it is; unlike that of scarlet fever. Its border



FIG. 128.—ERYSIPELAS BEGINNING ON THE NECK.

Child of 7 weeks, in the Children's Ward of the University Hospital in Philadelphia, suffering from chronic gastrointestinal disturbance. Improved slowly during 2 months. Then developed fever and eruption of erysipelas on the neck, with large bullae. Died after an illness of less than a week.

is sharply defined, elevated, and either clean cut or with irregular projections jutting out here and there into the healthy skin. When the process is severe vesicles or bullae form upon the affected skin (Fig. 128). Within 2 to 3 days the inflammation begins to disappear in the part first involved, and desquamation follows, usually in fine scales, but coarser and in larger pieces if vesicles have been present. In the meantime the border of the infiltrated region has extended more or less rapidly, sometimes a considerable area becoming involved in a few hours, and sometimes the advance being very slow.

The manner of the spreading and the degree of swelling depend largely upon the locality. Where the skin is firmly adherent to underlying structures, as at the chin, the patellæ and the condyles, the disease often passes around, leaving these areas unaffected. Where the tissue is loose, as at the genitals and the eyelids, the edema is great. When

¹ *Loc. cit.*

the disease starts at the nose this organ swells rapidly and the rash generally quickly spreads to the cheeks in the well-known butterfly form. It often stops here, but it may involve the eyelids, closing them for days, or may extend over one or both sides of the face and to the ears (Fig. 129). The whole scalp may be finally involved. Under such circumstances the head and face seem twice their natural size and the child is entirely unrecognizable. The inflammation may cease here, or may spread to the body. Developing about a vaccine pustule or other lesion on the arm or leg, it may remain confined to this locality, or may extend rapidly over the whole limb and thence to other parts, but not so often to the head as elsewhere. In some cases the disease appears to start on the mucous membrane of the nose, throat, or mouth and spread to the face, the first symptoms being a severe angina or coryza. In other cases it first attacks the mucous membrane of the vulva and extends to the thighs and other regions.



FIG. 129.—ERYSIPELAS WITH GREAT SWELLING OF THE HEAD.

Child of 14 months, in the Children's Hospital of Philadelphia. Disease began in the face, involved the head, closing the eyes, thence spread to the rest of the body. Death 9 days after the onset.

The tendency of the rash to spread varies greatly. It may be very slight, the dermatitis reaching but little beyond the point of original appearance. On the other hand the eruption may wander more or less rapidly over the whole body (*erysipelas migrans*), returning and attacking again parts from which it had disappeared but a short time before. This spreading is often in the form of repeated short advances, with intermissions during which improvement in the general symptoms leads to the false hope that the attack is over.

Symptoms Attending the Eruption.—The temperature as a rule rises very rapidly and remains at 104°F. (40°C.) or more, with but slight morning fall, as long as the spread of the rash is uninterrupted (Fig. 130). In very mild cases fever may be entirely absent or undiscovered. It generally is in proportion to the severity of the cutaneous manifestations. Each temporary cessation of the spreading of the disease, with subsequent recrudescence, usually is indicated by a fall of temperature followed by a rise (Fig. 131). An intermittent extension has therefore

an intermittent temperature; a steady spread, a temperature more continuously elevated. The lymphatic glands in the neighborhood of the dermatitis are nearly always inflamed. In severe cases the appetite is poor, the tongue dry, the pulse weak and respiration sometimes dyspneic. Restlessness, delirium or sopor not infrequently occur. The blood shows a leucocytosis most marked in the severe cases. Transient albuminuria

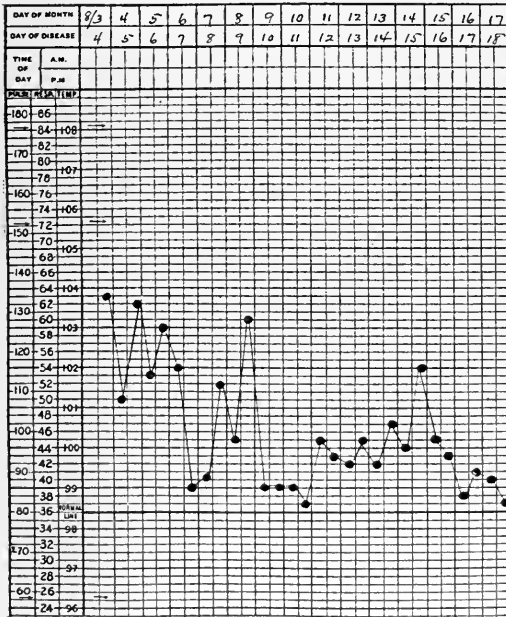


FIG. 130.

FIG. 130.—ERYSIPELAS, AVERAGE CASE.

Elsie S., aged 3½ years. Aug. 3, 4th day of disease, eruption spreading over left leg; Aug. 5, involving foot; Aug. 6, no extension; Aug. 8, rapid extension to buttock with increase of fever; Aug. 13, fresh extension, involving right leg and trunk; Aug. 14, development of bullæ, with increase of fever; Aug. 17, convalescing.

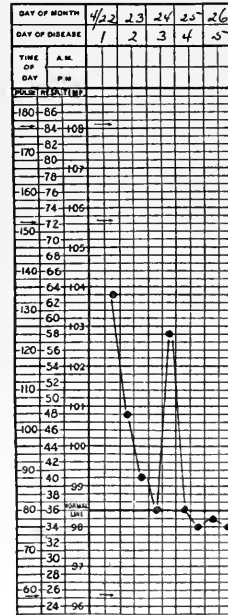


FIG. 131.

FIG. 131.—MILD ERYSIPELAS WITH RECRUDESCENCE OF FEVER ATTENDING EXTENSION OF RASH.

Edith S., aged 7 years. Apr. 22, red flush right cheek, extending over nose; Apr. 23, large blebs on right cheek, eye nearly closed; Apr. 24, extension to left cheek with renewed fever; Apr. 26, improving.

is occasionally present. The complex of symptoms varies, being often influenced by the complications which are prone to occur (Figs. 132 and 133).

The duration of the disease is extremely variable. As already stated the height of the affection in any one spot is reached in 2 to 3 days, and recovery in that region is rapid. An average duration of the entire attack is 7 to 9 days but it may last less than this or often much longer, and in cases of erysipelas migrans may continue occasionally even for months.

Erysipelas in Early Infancy.—This condition, and especially that denominated *Erysipelas neonatorum*, differs somewhat from the disease as seen later. The regions first attacked are oftenest the umbilicus, vulva, anus,

and the lesions of circumcision or vaccination, rather than the head. The onset is more insidious, and the child is restless and fretful, but often without fever, for some hours or even days, and sometimes at any period. When fever develops the spleen enlarges, vomiting and diarrhea are likely to occur and the dermatitis spreads widely. Loss of strength is

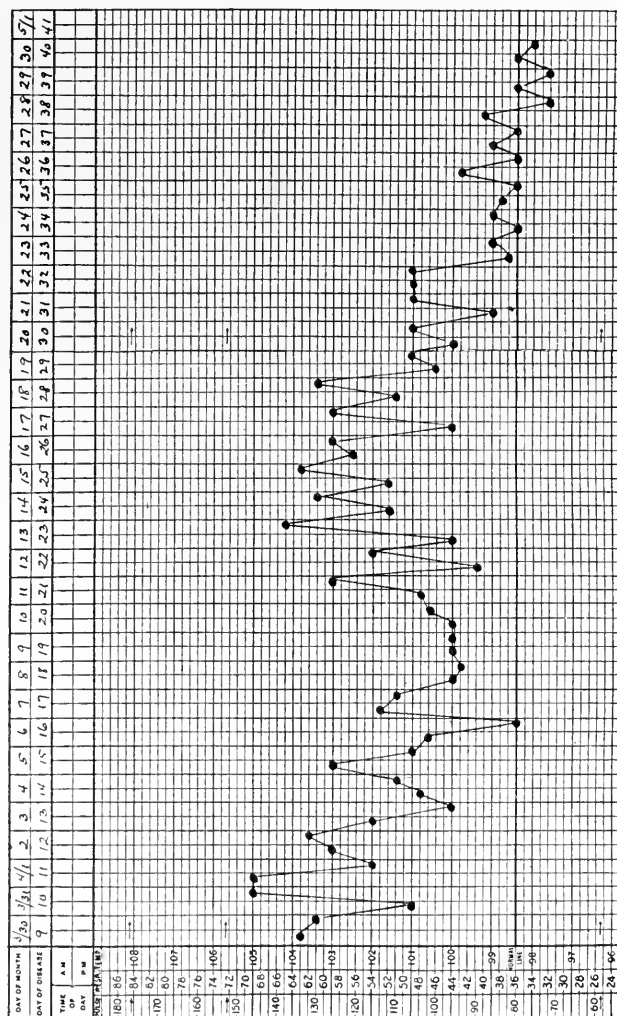


FIG. 132.—ERYSIPELAS WITH MULTIPLE CUTANEOUS ABSCESES.

John B., aged 3 years. Onset with vomiting, fever, and redness and swelling of face. Then developed meningitic symptoms, with delirium, retraction of head, rigidity of limbs. Lumbar puncture negative. Moderate leucocytosis. First abscess formed on head on the 11th day. Eruption extended to upper extremities and trunk, with formation of numerous subcutaneous abscesses here, and fresh ones on the head. Pus contained streptococci. Emaciation and weakness great. Slow disappearance of meningitic symptoms. Final recovery.

rapid septic symptoms appear, the pulse is very rapid and weak, food is refused, collapse supervenes, convulsions may occur, and death may follow within a week or even a day or two from the onset. There is a decided tendency for the disease to assume the wandering type if the infant lives long enough. The eruption differs little from that seen later in life, except that the swelling is liable to be greater and more tense, the redness less intense, the boundary less raised and desquamation more liable to occur. The cases which recover generally develop cutaneous abscesses.

Complications and Sequels.—Suppuration in the subcutaneous tissues is not infrequent (Fig. 132), and necrosis of portions of the skin have been reported. Bronchitis, pneumonia (Fig. 133), peritonitis and endocarditis may occur, and edema of the larynx sometimes develops. Meningitis is an occasional complication, although many subjects exhibiting its symptoms show no such lesion at autopsy. Nephritis is not common. Suppuration of joints or of lymphatic glands is sometimes seen.

Erysipelas may occur in combination with many other diseased conditions, especially, as indicated, with wounds. It may be a complica-

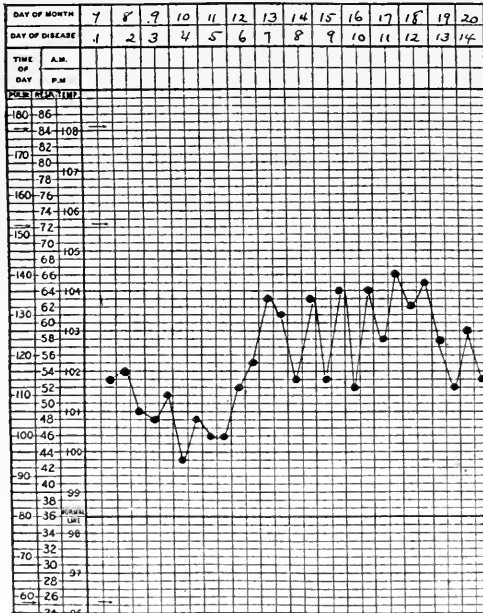


FIG. 133.—ERYSIPELAS, SEVERE CASE. BRONCHOPNEUMONIA.

Adelaide C., aged 7 months. Feb. 7, swelling of left labia; Feb. 14, involvement of right labia; Feb. 16, bronchopneumonia; Feb. 20, rash has been steadily spreading, involving abdomen and lower extremities; severe toxic state; death.

tion of other infectious disorders, as scarlatina, malaria, typhoid fever, cerebrospinal fever and diphtheria. In the last three it generally results from the development of a bed sore or other open wound.

Relapse and Recurrence.—In addition to the frequent recurrences, with pseudo-crises so characteristic of the disease, true relapse may show itself after several days or even a few weeks. It probably owes its origin to the remaining of small infiltrated, infected foci in the skin. The relapse may be repeated a number of times.

Recurrence in the sense of a new infection is more common than relapse. In fact, one attack creates absolutely no immunity, but rather seems to predispose to later ones. Frequent recurrence is liable to result in permanent thickening of the affected areas, especially the scrotum and the eyelids.

Prognosis.—One of the most important factors is age. In the new born erysipelas is almost always fatal and at any period of infancy it is serious, especially in the first 2 or 3 months of life. In childhood

recovery generally takes place if the disease is uncomplicated and of moderate extent.

The situation involved is of importance as well. The prognosis is prone to be worse if the head is attacked. Wandering erysipelas has a graver prognosis than the more limited form, since its long duration tends to produce exhaustion.

Among unfavorable symptoms are very high temperature, weakness of the heart, and cerebral symptoms. All complications increase the gravity of the case and death may follow pneumonia and sometimes peritonitis, meningitis, and involvement of the kidneys.

Diagnosis.—This rests upon the combination of constitutional symptoms with a dermatitis of peculiar nature, often with vesicles and with marked tendency to extension. The latter features and the sharply defined border differentiate it from other forms of dermatitis. The rash of scarlet fever is distinguished by its punctiform character. Deep-seated phlegmonous inflammation lacks the characteristic border, as does also lymphangitis, which has, too, an entirely different outline, since it follows the course of the lymphatic vessels. Erythema infectiosum, while having a distribution and color upon the face suggesting erysipelas, lacks the sharp outline and the infiltration, and exhibits a morbilliform rash on the trunk and limbs.

Treatment. Prophylaxis.—Especial care should be taken to insure asepsis of the umbilical wound of the new born. Infants should be removed from their mothers if the latter are suffering from puerperal fever. All possible precautions should be employed in performing vaccination and circumcision, and all children with open wounds should be carefully separated from patients with erysipelas.

Treatment of the Attack.—So far as drugs are concerned, although the number tried is very great, none can be called specific. The internal administration of tincture of the chloride of iron in large doses has long been a favorite. There appears to be some evidence that it does good, and at any rate it can be said for it that it can do no harm. Quinine and salicylic acid in full doses are each favorite remedies with many. Locally the remedies most in favor are petrolatum, ointment of ichthyol (5 to 15 per cent. or stronger); ointment or glycerin-solution of resorcin (30 per cent. or more); powders of iodoform, oxide of zinc, or starch and salicylic acid; compresses or solutions of bichloride of mercury (1:2000), boric acid, lead water and laudanum, and alcohol and water. Of all of these the application of ichthyol ointment or of ichthyol collodion (10 per cent. or more) is perhaps one of the most popular, and the course of the disease often appears to be much abbreviated by the treatment. Hypodermic injection of anti-streptococcic serum or of autogenous vaccines has been employed, but the results have been inconclusive. I have seen prompt recovery follow their use, but could not determine that this was because of it. Erdman's¹ experience in 800 cases of erysipelas did not show any shortening of the attack in 95 instances in which vaccines had been used.

Other treatment is symptomatic. The general strength, and especially that of the heart, may require alcoholic stimulants and digitalis. Suitable nourishment must be given; excessive temperature may require warm or cool bathing; threatening nervous symptoms may be treated hydropathically or may need bromides, antipyrine, or occasionally, opium. Complications call for treatment appropriate to them.

¹ Journ. Amer. Med. Assoc., 1913, LXI, 2048.

CHAPTER XII

DIPHTHERIA

History.—Recognition of an infectious epidemic disorder characterized by the development of a membrane-like coating generally situated in the fauces, nose or larynx, appears to date from very early times. Perhaps the first clear account was given by Aretaeus¹ in the 1st century. It ravaged Europe in the 16th and 17th centuries and was described in America by Douglas² in 1736 and very completely by Bard³ in 1771. Variouslly designated, it was first called "Diphtheritis" (*διφθερα* = a membrane or skin) by Bretonneau.⁴ Since the discovery of the specific bacillus by Klebs the term diphtheria is properly applied only to the disease dependent upon this germ. The other pseudomembranous affections of the throat should be entitled pseudodiphtheria.

Etiology. Predisposing Causes.—The disease is of very frequent occurrence, and prevails in all climates and all civilized countries without regard to locality, although perhaps most common in cold, damp regions and in the cooler months of the year. Humidity is a decided factor through its influence in favoring catarrhal affections, any acute or chronic catarrhal disorder of the mucous membrane of the nose or throat, as also the presence of adenoids and of enlarged tonsils, rendering the subject more susceptible. Poor sanitation or any sort, and impaired health in general, increase the tendency to the disease by diminishing the resisting powers. For this reason, certain other infectious disorders, especially scarlet fever and measles, as well as influenza and pertussis, augment the susceptibility.

Age is an important predisposing factor. The disease is most frequent up to 10 years, and especially from 1 to 5 years of age. Of 9011 cases admitted to the Philadelphia Hospital for Contagious Diseases (Welch and Schamberg),⁵ 4076 were from 1 to 5 years old. It is less common in the 1st year and especially in the first 6 months. In 2600 cases reported by Rolleston,⁶ there were but 20 occurring in the 1st year; and in 2711 cases in children Baginsky⁷ found only 15 (0.15) per cent.) under 6 months of age. Yet new-born children are occasionally attacked (Jacobi)⁸ (Riesman,⁹ infant of 11 days; Stimson,¹⁰ 10 days; primary nasal). There exists often a very striking family predisposition, but apart from this the *individual susceptibility* is not very great and the disease much oftener limits itself to a single child in a family than is the case in some other infectious diseases, notably measles. This immunity (see p. 460) may be overcome through various influences, and as a result the disease may appear at times in extensive epidemics in some years, with but few cases in others. In large cities it is *endemic* to a varying degree, and it may develop sporadically in localities where its origin cannot be traced.

¹ De causis et signis acut. morb. L. I, Cap. 9. Ref. Baginsky in Nothnagel's Handb. d. spec. Path. u. Therap. Diphtheria.

² Pract. Hist. of a new erupt. miliarial fever with angina ulcerculosa, Bost., 1736. Ref. Baginsky, *loc. cit.*, 6.

³ Transac. Amer. Philosoph. Soc., 1779, I, 338.

⁴ Des inflam. spec. des tissu muqueux etc., Paris, 1826. Ref. Baginsky, *loc. cit.*

⁵ Acute Contagious Diseases, 1905, 611.

⁶ Amer. Jour. Dis. Child., 1916, XII, 47.

⁷ *Loc. cit.*, 52.

⁸ 20th Cent. Pract. Med., XVII, 77, Article Diphtheria.

⁹ Phila. Med. Jour., 1898, March 5.

¹⁰ New York Med. Jour., 1907, Dec. 14.

Exciting Cause.—This is now well recognized to be a specific bacillus first recognized by Klebs¹ in 1883, and shown to be the sole cause of the disease by Loeffler² in 1884. Roux and Yersin³ in 1888 proved that this germ was capable of occasioning in animals the same paralytic conditions as are seen in man, and demonstrated the production by it of a poisonous substance upon which the various symptoms of the disease depend. All later observations confirm the etiological relationship of the germ.

The microorganism is a non-motile, non-spore-bearing Gram-positive bacillus, averaging about the length of the tubercle bacillus but thicker. It is straight or slightly curved and often somewhat club-shaped at the ends. It has the characteristic peculiarity of varying greatly in size, form and staining qualities, depending on its age and on the culture-medium employed, and stains well with alkaline methylene blue as recommended by Loeffler, but always with unstained spots, the club-shaped ends being most intensely colored.

Life History.—The bacillus is readily killed by a temperature of 58°C (136.4°F.) but is not affected by cold. Under normal conditions it is very tenacious of life. In the great majority of cases it disappears from the throat in 2 to 4 weeks from the beginning of the disease, but it may sometimes persist for weeks or months in a virulent form in the throat or nose of those who have passed through an attack or who have merely been exposed, or it may live long in various objects entirely apart from the body. Abel⁴ found it living for over 6 months on toys which had been kept in a dark place, Klein⁵ for 18 months in cultures, Le Gendre and Pochon⁶ for 15 months in the throat, and Valagussa⁷ living but completely dry for 26 months. It occurs sometimes in the secretion from purulent otitis media or may occasionally be found in the tissues of the body or the blood and the urine. Thus in 209 fatal cases Councilman, Mallory and Pearce⁸ discovered it in the heart's blood 12 times, in the liver 42 times, and in the spleen 26 times; frequently associated with the streptococcus, less often with the staphylococcus or the pneumococcus. Sommerfeld⁹ discovered it in the blood in 42 out of 320 cases (13.1 per cent.). It has also been found in the lungs, bone-marrow, kidneys and lymphatic glands. The diphtheria germ is, however, as a rule not widely distributed in the body, being confined in most cases to the pseudomembrane and the surface of the mucous membrane.

The disease is *primarily a local one*, the germs being deposited and growing upon a mucous membrane which was not in a healthy condition. Some abrasion, although slight, is necessary as the original nidus. The symptoms resulting depend chiefly on the absorption of the poisonous principle produced by the bacillus, and perhaps partly upon the action of associated germs especially the streptococcus pyogenes and the staphylococcus pyogenes. There is no diphtheria without the specific germ, and it is equally true that the mere discovery of this on the mucous membrane does not indicate the presence of diphtheria.

Diphtheria bacilli may lose their virulence, and such germs, although

¹ Verhandl. d. Cong. f. inn. Med., 1883, II, 139.

² Verhandl. d. Cong. f. inn. Med., 1884, III, 156.

³ Annales de l'institute Pasteur, 1888, II, 629.

⁴ Centralbl. f. Bakt., 1893, XIV, 756.

⁵ Centralbl. f. Bakt., 1890, VII, 492.

⁶ Bull. soc. des hôp., 1895, XII, 815.

⁷ Riv. di clin. pediat., 1909, VII, 332.

⁸ Bact. and Pathology of Diphtheria, 1901, p. 16.

⁹ Arch. f. Kinderh., 1913, LX-LXI, 698.

morphologically and in reality identical with the virulent ones, are without danger to those acquiring them. As a rule, the more virulent the germ, the worse the attack of the disease; but to this there are many exceptions, and the mildest cases may transmit very virulent germs and occasion a severe attack in another subject.

Another organism has been described, known as the pseudo-diphtheria bacillus, or the Hofmann-Wellenhof¹ bacillus, which is very similar to, but not identical morphologically and culturally with that of diphtheria, and is not capable of producing the disease. Whether this is, in reality, different from the avirulent Klebs-Loeffler's bacillus, or only a variant, is not yet positively determined, but the latter seems very possible.

Transmission.—The disease is a strictly *infectious* one and never develops independently. Indirect transmission is much the least frequent method of communicating the infection. It may occur through infected milk, or occasionally by domestic animals, or by books, toys, clothing and other inanimate objects. In the large majority of cases the germs are communicated directly, either from an unrecognized case, or by a convalescent, or by a healthy person who has never had diphtheria but who has the virulent bacteria present in the throat or nose. Such convalescents and healthy persons are called "carriers." The remarkable persistence of the germs on the mucous membranes of convalescents or of healthy persons which is frequently seen, and their tenacity of life, account for cases which occur sporadically and without discoverable source. The existence of mild unrecognized nasal diphtheria is a very fruitful cause of dissemination of the disease in schools, hospitals, and elsewhere. Transmission may occur from the beginning of the attack or even before any symptoms appear. It continues possible as long as the bacilli remain virulent in the carrier. The difficulty in controlling the spread of the disease is shown by the investigations of the Massachusetts Boards of Health² according to which at least from 1 to 2 per cent. of all dwellers in cities have genuine diphtheria bacilli on the mucous membrane of the throat, as have from 8 to 50 per cent. of those who have been in any way exposed to the disease. Pennington³ found that 10 per cent. of all the apparently healthy children examined in the public schools of Philadelphia exhibited diphtheria bacilli, non-virulent in about $\frac{1}{2}$ of the cases. von Sholly⁴ discovered the germs in the throats of 5.6 per cent. of 1000 healthy school-children; and Schrammen⁵ in 6.5 per cent. of 704 school-children, although at the time there was not a single case of diphtheria in the schools or in the families of the children.

Pathological Anatomy.—The lesions of diphtheria may be divided into primary and secondary. The *primary lesion* consists in the production of the pseudo-membrane. This is found oftenest upon the tonsillar tissue in the fauces and the nasopharynx; very frequently in addition upon the pharynx, uvula, larynx, trachea and bronchi; less often in the mouth, vulva, vagina, middle ear, conjunctiva, esophagus, stomach and intestine. It may also occur occasionally on wounds, as upon the penis after circumcision. Macroscopically the pseudo-membrane is a greyish-white or yellowish-white substance, tough or friable, covering a

¹ Wien. med. Wochenschr., 1888, XXXVIII, 60.

² Journ. Mass. Assoc. Boards of Health, 1902, July. Ref., Welch and Schamberg, Acute Contagious Diseases, 1905, 619.

³ Journ. Infect. Dis., 1907, IV, 36.

⁴ Research Lab., Dept. of Health, New York City, 1905, I, 88; Journ. Infect. Dis., 1907, IV, 337.

⁵ Centralbl. f. Bakt., Orig., 1912-13, LXVII, 123.

smaller or larger area. In places it can be removed rather readily, but for the most part only with injury to the tissues beneath. On the larynx it is firmly adherent; on the trachea easily detached. In severe cases it soon assumes a dark-greenish or blackish color or a gangrenous appearance. The surrounding tissue is deeply congested.

Microscopically the pseudomembrane consists of a degeneration and necrosis of the mucous membrane combined with an inflammatory fibrinous exudate from the underlying blood-vessels. This exudate may be in granular form, but oftener appears as a fine network which includes in its meshes leucocytes and degenerated epithelial cells as well as diphtheria bacilli and micrococci. The blood-vessels and connective tissue beneath the pseudo-membrane exhibit thickening and hyaline transformation. The diphtheria bacilli do not reach the lowest layers of the exudate.

A distinct pseudo-membrane is not necessarily present in diphtheria. Not infrequently there appear instead merely a few spots of yellowish, soft secretion in the crypts of the tonsils (*diphtheritic folliculitis*), while in other cases only a catarrhal condition of the mucous membrane of the throat is discoverable (*catarrhal diphtheria*). Nevertheless, even in the latter condition, the characteristic necrotic changes can be discovered in the superficial cells.

The primary lesion of diphtheria possesses little importance except when in the larynx, where by its mechanical interference it may cause suffocation. It is the *secondary lesions* which constitute the chief danger of the disease. These are the widespread degenerative changes in the various tissues of the body, the result of the action of the powerful toxin produced by the diphtheria bacillus in the primary lesions and absorbed and distributed by the blood and the lymph. Other microbes present in the pseudo-membrane probably aid in the destructive process, either by the local formation of toxins and the subsequent absorption of these, or by directly entering the blood-vessels and producing a septicemia; but the chief danger is from the toxin of the diphtheria bacillus itself. The changes consist in cellular degeneration in all the organs. The cervical, bronchial, and mesenteric lymphatic glands are enlarged and congested and show leucocytic infiltration and often hemorrhages. Scattered bronchopneumonic areas are common, found in over one-half of the autopsies. A serous or sero-fibrinous pleurisy may occur. Small hemorrhages may be present in the skin, pleura, pericardium and endocardium and beneath the capsule of the liver and spleen. The cardiac muscle suffers degeneration in all long-continued fatal cases. The arteries sometimes exhibit proliferation in the endothelial lining. The spleen is increased in size, soft, and dark-red; its follicles enlarged, cellular degeneration prominent and hemorrhages frequent. The kidneys are larger than normal, soft, and exhibit changes varying from simple degeneration to a serious degree of acute nephritis, there being no type of nephritis peculiar to diphtheria (Councilman, Mallory and Pearce).¹ The interstitial and glomerular forms are more common in older children and in long-continued cases. The liver is slightly enlarged and soft, and with necrotic alteration of the cells, especially in the interior of the lobules. The brain and spinal cord are generally little altered. Occasionally hemorrhages or a moderate fatty degeneration of the white substance is found. The cranial and spinal nerves exhibit a wide-spread degeneration.

¹ *Loc. cit.*, 152.

Symptoms.—The symptoms of diphtheria vary greatly with the locality affected, as well as with the intensity of the intoxication and the complications which may arise. A general description may be given of an average case of the commoner form, faucial diphtheria, as illustrating the ordinary type of the disease.

ORDINARY TYPE. FAUCIAL DIPHTHERIA. Incubation.—This is certainly short, varying generally from 1 to 4 days. The difficulty in determining it exactly depends on the uncertainty as to the length of time the bacilli may have been living upon the mucous membrane without producing local or general infection.

Invasion.—Symptoms of invasion are by no means characteristic. Although the process is primarily local, the first clinical manifestations noticed are more constitutional in nature, and consist in chilliness, slight fever, and loss of appetite; sometimes vomiting, headache, and swelling of the cervical glands, and occasionally convulsions. There may or may not be complaint of sore throat. Inspection shows only slight coating of the tongue, an irregular redness and swelling of one or both tonsillar regions, and often a dark-red coloring of the mucous membrane of the mouth.

Symptoms of the Attack.—Within 24 hours, however, the appearance is more characteristic and a small or larger yellowish-white or grayish-white deposit is seen, which resembles the secretion of ordinary follicular tonsillitis, but which is removable, as a rule, only with difficulty, and appears to be of a more membrane-like character. New membrane generally forms rapidly after such removal. Sometimes the early appearance of the deposit is more gelatinous, the tonsil seeming to be covered by a slightly cloudy mucilaginous secretion.

The pseudo-membrane spreads, and, if limited to one tonsil at the outset, extends to the other in from 2 to 5 days and often to the uvula, the pillars of the palate and the soft palate. Its color has, in the meantime, become a dirty grey, the odor from the mouth is very offensive, the swelling has much increased, and by the end of the 1st week the entire fauces, and to some extent the pharynx, may be covered by a thick pseudo-membrane, sometimes almost closing the throat, the nose being also, as a rule, involved to some extent. (See Nasal Diphtheria, p. 449.) The degree of difficulty in swallowing, and the amount of pain in the throat are very variable and do not seem to bear any relation to the extent of the inflammation.

Meanwhile the constitutional symptoms increase gradually in severity. The cervical glands grow larger, generally in proportion to the degree of faucial inflammation. The *temperature* exhibits no exact relation to the severity of the symptoms and is usually about 101° to 102° F. (38.3° to 38.9° C.); this comparatively low degree being characteristic of the disease (Fig. 134). Sometimes, however, it reaches 103° or 104° F. (39.4° to 40° C.) or more. It is generally highest at the beginning of the attack and then falls somewhat rapidly or slowly, although renewed rises may occur as the disease extends to new regions of the throat or as complications develop. Finally it reaches normal by lysis as the local symptoms improve. The *pulse* is generally weak and much more rapid than is in accord with the elevation of temperature, and the blood-pressure is low. This disproportion of the pulse-rate to the temperature is a very characteristic symptom. The appetite is lost, vomiting is common, constipation is the rule. The *urine* exhibits albumin in the majority of cases, although generally in not large amount, and epithelial

cells and hyaline casts in many instances. The knee-jerks are often absent.

The *blood* shows a marked polymorphonuclear leucocytosis, which increases as the disease advances and diminishes during convalescence. The percentage of red blood-corpuscles and of hemoglobin is reduced in all severe cases. One of the most marked symptoms of the disease is the *prostration* which the diphtheria toxin produces, frequently out of all proportion to the severity or duration of the other symptoms. Increasing anemia, loss of strength, and emaciation may be decided. Slight delirium may be present at the outset, and decided apathy, somnolence, or irritability later.

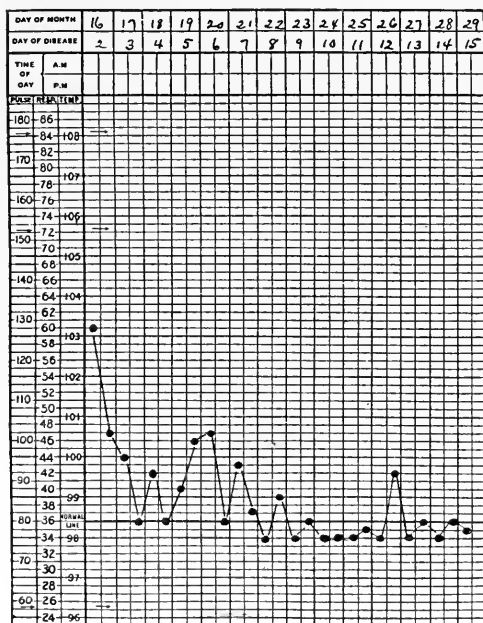


FIG. 134.—TONSILLAR AND NASAL DIPHTHERIA.

Mary K., aged 9 years. Feb. 15, onset with nausea, vomiting, sore throat, and fever; Feb. 16, tonsils and arches covered, nasal discharge; Feb. 18, great improvement; Feb. 21, throat clean. Given 7500 units antitoxin on 16th; 7500 on 17th; 6000 on 18th; 5000 on 19th. Philadelphia Hospital for Contagious Diseases. (Courtesy of Dr. B. F. Royer.)

About the end of the 1st week, or frequently not until later, improvement may begin, indicated by softening and separation of the pseudomembrane and the gradual amelioration of symptoms. The disappearance of the pseudomembrane is often very rapid, especially under serum-treatment. Occasionally deep ulceration of the mucous membrane then becomes visible. Very often, however, the local and general symptoms continue a much longer time. The albuminuria generally persists until the end of the 2d or 3d week.

Convalescence is usually slow and debility long-continued. Weakness of the heart is a very prominent symptom even at this period and death may occur from this cause after the membrane has nearly or quite disappeared and the child has seemed to be out of danger.

Very frequently associated with faucial diphtheria is involvement of the nasopharynx. This decidedly modifies the course and duration of the disease. So also laryngeal diphtheria may result from a spread of the membrane from the fauces to the larynx. The symptoms of the two affections will be described separately.

NASAL DIPHTHERIA.—Nasal diphtheria may be primary in the nose and either remain limited to this region or may spread to the nasopharynx, the throat, or the larynx. In other cases the nasal involvement is secondary to the affection in the fauces or nasopharynx.

Involvement of the nose and nasopharynx as a *secondary* affection is common and adds to the severity of the case and the discomfort of the patient. Respiration through the nose becomes difficult or impossible, and an offensive and very irritating discharge flows from the nostrils, causing excoriation and swelling of these and of the upper lip. Membrane may be seen lining the anterior nasal passages, and epistaxis is frequent. Constitutional symptoms in these cases are generally severe, and the duration of the attack is prolonged. The disease may extend to the ear, producing a purulent otitis media as a complication and resulting in a still longer continuance of the fever and of other symptoms.

Primary nasal diphtheria is commonest in infants, very frequently remains for some time undiscovered, and is a fertile source of contagion. When confined to this region either no general symptoms at all are noticed, or there is only slight fever and malaise. The child appears to have merely a cold in the head, but the character of the thin and very irritating nasal discharge is suspicious. Examination of the anterior nares will now often reveal the presence of pseudomembrane and of diphtheria bacilli. These cases run a somewhat chronic course. In other instances the affection spreads to the nasopharynx and throat, and the constitutional and local symptoms are decided. Diphtheria *primary in the nasopharynx* is generally severe.

Nasal diphtheria, either primary or secondary, is a common form of involvement. In 1962 cases of diphtheria in the Boston City Hospital, Burrows¹ estimated that the nose exhibited the disease in about 40 per cent., and in 1200 cases of faucial diphtheria, Rolleston² found involvement of the nose in 41.6 per cent.

LARYNGEAL DIPHTHERIA.—This may properly be called *laryngo-tracheal diphtheria* since the pseudomembrane may involve a portion of the trachea as well, or even extend into the bronchi. It is one of the most dangerous forms, most of the deaths in diphtheria depending upon this cause. The vast majority of cases of pseudomembranous laryngitis, or "true croup," are of a diphtheritic nature, and almost always secondary to lesions in the nose or the throat, although they often appear primary on account of these lesions being very slight, or transitory and overlooked. Diphtheria of the larynx develops about the 4th or 5th day of the disease. The first symptoms are hoarseness, a ringing cough and a very slightly noisy and prolonged respiration. Apart from the influence of any accompanying pharyngeal involvement there is little, if any constitutional disturbance; and fever may or may not be present (Fig. 135). The symptoms are, in fact, local and are those of laryngeal stenosis. Generally they come on very rapidly, being fully developed by the 2d day of laryngeal involvement. The voice becomes very hoarse,

¹ Amer. Jour. of the Med. Sciences, 1901, CXXI, 125.

² Report Metropolitan Asylums Board, 1906.

whispering or absent; the cough peculiarly metallic; and dyspnea severe, both inspiration and expiration being prolonged and labored. The child has an extremely anxious expression; is restless and tossing; often sits upright in bed with head thrown back and draws its breath only with difficulty and with the aid of all the accessory respiratory muscles and with spreading of the alæ of the nostrils. There is retraction of the epigastrium and the supraclavicular and suprasternal spaces and a lifting of the thorax high with every inspiration. Cyanosis is marked and the skin is clammy. Laryngoscopic examination reveals edema of the mucous and sub-mucous tissues and the presence of pseudomembrane in the larynx; sometimes one predominating and sometimes the other.

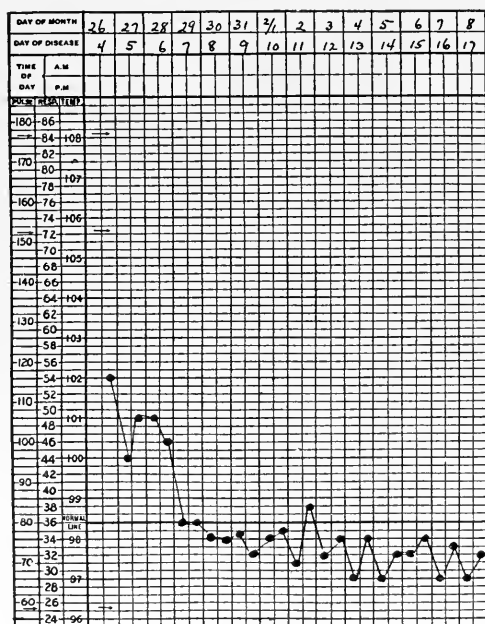


FIG. 135.—TONSILLAR AND LARYNGEAL DIPHTHERIA.

Joseph S., aged 3 years. Jan. 23, onset with fever and croup; Jan. 26, both tonsils covered, complete loss of voice, some stenosis; Jan. 27, less pseudo-membrane and stenosis; Jan. 29, throat clean, a trifle croupy. Treatment, Jan. 26, 10,000 units; Jan. 27, 10,000 units; Jan. 28, 5000 units. Philadelphia Hospital for Contagious Diseases. (Courtesy of Dr. B. F. Royer.)

The symptoms vary from time to time, now being much less marked; now increasing to the point of impending suffocation. The child then becomes livid, grasping at its throat or at the air with its hands, almost ceasing to breathe, and being apparently at the point of death. This change in the picture depends upon the varying degree of edema present. Occasionally relief is afforded by the coughing up of portions of pseudomembrane, but the deposit is soon reproduced and the threatening symptoms return. Before the days of antitoxin-treatment the majority of cases grew worse with varying rapidity, respiration becoming feeble, cyanosis increasing, the heart failing, a stuporous condition developing sometimes with convulsions and death taking place after 24 to 48 hours or sometimes not for several days or a week. In other instances the child

dies suddenly from lack of air in one of the suffocative attacks. In the cases which recover, relief, at first partial and then permanent, may follow the coughing up of membrane, or the stenosis may gradually disappear.

Laryngeal diphtheria is a frequent form of the disease. In the 1962 cases of the Boston City Hospital, reported by Burrows,¹ laryngeal stenosis occurred in 17 per cent.

Diphtheria may be classified also according to the character of the symptoms, the severity, and the degree of constitutional infection.

MILD DIPHTHERIA.—Not infrequently the disease is of so mild a nature that it is either entirely overlooked or a positive diagnosis made possible only by bacteriological examination. Constitutional symptoms are nearly or entirely absent. As already stated primary nasal diphtheria is often of this nature, for unless membrane can be discovered—and it is not always present—the diagnosis is entirely bacteriological, since the profuse nasal discharge, although severe, could readily depend upon some other affection.

In other cases there are only the symptoms of a lacunar tonsillitis, the child suffering from sore throat and some degree of fever, but not from any constitutional depression (Fig. 136). Should an examination of the throat chance to be made some of the tonsillar crypts are found filled with secretion exactly as in ordinary lacunar tonsillitis. This deposit may disappear in 24 hours or less and leave the patient feeling completely well. Diphtheria bacilli will, however, be found in the throat on bacteriological examination. Sometimes these cases persist as apparently simple lacunar tonsillitis for a few days and then develop membrane and run the ordinary course of diphtheria. In other cases a rapid recovery of the tonsillitis is followed in a few days by the development of laryngeal diphtheria, and in still others the diphtheritic lacunar tonsillitis is rather persistent and is accompanied by a degree of constitutional depression out of all proportion to the lesions seen in the throat, although it may perhaps not be sufficient to confine the patient to bed.

CATARRHAL DIPHTHERIA.—This term is applied to the mild cases in which there is an entire absence of membrane in either the nose or throat, the lesions being apparently simply those of a catarrhal inflammation. Constitutional symptoms are absent or insignificant. Diphtheria bacilli are, however, found on bacteriological study, and histological examination shows the characteristic pathological changes in the epithelium of the mucous membrane. As in lacunar diphtheria the disease may give rise to laryngeal involvement.

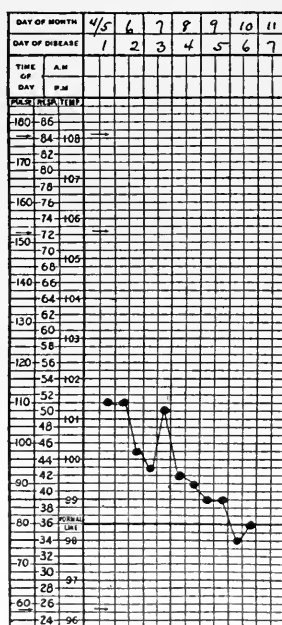


FIG. 136.—MILD TONSILLAR DIPHTHERIA.

J. A. S., aged 4 years. Deposit limited to tonsils, yellowish white, slight submaxillary swelling. General condition good. Slight albuminuria. 3000 units antitoxin given on Apr. 8, although symptoms already improving.

¹ *Loc. cit.*

SEVERE FAUCIAL AND NASOPHARYNGEAL DIPHThERIA.—As laryngeal diphtheria is always a most dangerous affection, only the cases of severe faucial diphtheria are considered in this connection. Many of these are well called "*septic diphtheria*," the general symptoms being those of profound sepsis, the streptococci appearing to share with the diphtheria bacilli in the poisoning of the patient. Others owe their severity entirely to the action of the diphtheria toxin. These grave cases may begin as ordinary ones and then steadily grow worse, or the onset may be sudden and severe. The membrane is widespread; the tonsils and adjacent parts greatly swollen, often assuming a gangrenous appearance; the mucous membrane of the nose shares in the process and secretes an abundant offensive mucopurulent fluid. Hemorrhage may take place from the mouth and nose; the lips are swollen, fissured and sometimes exhibit membrane, as does occasionally the lining of the cheeks; the tongue is thick and dry; the eyes are sometimes involved in the process; the cervical lymphatic glands are greatly swollen and often the periglandular connective tissue also. Respiration through the nose is much obstructed. Swallowing may be difficult and food regurgitated through the nose. The face is peculiarly pale and more or less cyanotic, and the surface of the body pale or sometimes exhibiting petechiæ or a dusky redness; the extremities are cold; the pulse feeble and rapid, or occasionally unusually slow. The heart shows evidence of increasing dilatation and loss of muscular tone, the sounds being weak and murmurs sometimes present. The temperature is variable, sometimes low, often high, and subject to marked fluctuations. Abdominal pain, vomiting, and diarrhea are common and prostration extreme. The mind may be clear or there may be restlessness or delirium or, oftener, profound apathy or stupor. The urine is frequently scanty and contains tube-casts and albumin in large amount; the spleen is enlarged.

The attack lasts a variable time. The development of the membrane reaches its height in 4 or 5 days, and no change in it may be apparent for several days more. At the end of about the 1st week it begins to disintegrate in favorable cases, and may disappear in a few days more. Sometimes, however, it persists for 2 weeks or longer. After its disappearance the mucous membrane is left raw-looking and sometimes ulcerated. The toxic symptoms reach their height with the maximum growth of the pseudomembrane, and although usually ameliorating as this disappears, by no means always do so, and in any event improve only slowly. A complicating laryngeal diphtheria is not likely to develop after the 1st week. The course is not usually much longer than a week in fatal cases, the child dying generally from increasing debility or bronchopneumonia; or more unexpectedly from uremic convulsions, heart-failure, or involvement of the larynx. Sometimes the fatal ending occurs only after improvement in local symptoms has commenced and convalescence seems assured; and may even be delayed until the 3d week of the disease or later.

Malignant diphtheria is a term applied to the very worst of the severe cases. The symptoms described are seen in a specially marked form. The pseudomembrane is widespread and the process sometimes necrotic or gangrenous; the toxemic symptoms are usually severe; the involvement of the cervical glands and cellular tissue great; and purpuric patches and hemorrhages from various mucous membranes may be present. Death occurs in 24 to 48 hours from the onset.

UNUSUAL LOCALIZATIONS.—Membrane may appear in unusual regions, either primarily, or secondarily to diphtheria of the throat or

nose. In very severe cases it may spread to the buccal cavity, lips, or the adjacent portion of the face. In one instance I observed it on the lining of the cheeks before it could be discovered in the throat, the primary lesion in this instance appearing to be on a circumcision wound. Diphtheria of the conjunctiva is rare. The disease may be primary or secondary upon the penis or the scrotum, producing great swelling, or upon the vagina and vulva. In the last the genitals are much infiltrated and a thin, irritating discharge excoriates the neighboring skin. Any cutaneous wound, especially that of tracheotomy, or even an eczematous area may be attacked by the diphtheritic process, especially in much debilitated subjects. Exceptionally it may be primary in the bronchi and spread thence to the larynx. Secondary involvement of the trachea and bronchi is common (Fig. 137); that of the tongue, esophagus, stomach and intestines rare.

Complications and Sequels.—One of the most frequent complications of diphtheria is *bronchopneumonia*. This is particularly liable to occur when the larynx is involved; in severe septic cases; and in infancy. It depends not only upon the presence of the diphtheria bacillus, but upon associated microorganisms, especially the streptococcus and the pneumococcus. It may develop at any time during the course of the disease and may be found at autopsy in over $\frac{1}{2}$ of the cases. Croupous pneumonia is only an accidental complication.

Cardiac failure is one of the most dreaded of complications or sequels. It results from degeneration of the cardiac muscles or ganglia, or an involvement of the pneumogastric nerve. Alteration of the bundle of His may or not be present. In other instances it is produced by the formation of a heart-clot. It may occur at any time during the course of the disease; even upon the 1st day in malignant cases; but is witnessed most frequently as a sequel in the 3d or 4th week during convalescence, and generally after severe attacks of diphtheria. If developing during the attack it takes place oftenest while the disease is at its height, or after the membranè has nearly or quite disappeared. The evidences of heart-failure may come on gradually, lasting 3 or 4 days or less, and consist in abdominal pain; obstinate vomiting; dyspnea; weak, compressible pulse which is usually rapid and irregular, sometimes slow; feeble heart-tones sometimes with faint "accidental" murmurs or a galop rhythm, and the percussion evidences of cardiac dilatation. The surface of the body is pale and the tissues may be dropsical. In other cases heart-failure is abrupt, the child dying suddenly when raised or when moving itself in bed, or, during convalescence, when walking about the room or even after undue excitement.

Sometimes repeated attacks of impending heart-failure occur extend-



FIG. 137.—UNUSUALLY WELL-DEVELOPED TRACHEAL AND BRONCHIAL CASTS.

From a case of diphtherial croup. (Lennox Browne, *The Throat and Nose and Their Diseases*, 1899, 5th Ed., 529.)

ing in all over a considerable period, to be followed finally by recovery or by a fatal ending. Accompanying this condition there is generally emaciation and loss of appetite and strength. Moderate disturbance of the heart, as shown by irregular and rapid pulse, faintness, and shortness of breath, is very common in diphtheria. White and Smith¹ found cardiac symptoms in 878 out of 946 cases of diphtheria. Occasionally permanent valvular lesions persist as sequels.

Nephritis is a frequent complication of diphtheria, evidenced by the occurrence of moderate albuminuria in from $\frac{1}{3}$ to $\frac{2}{3}$ of all cases, and in nearly every case at all severe. Baginsky² found albuminuria 417 times in 993 cases of diphtheria, and in 256 of these there were morphological elements present also. It depends upon degeneration of the renal epithelium through the action of the toxin. The urine may be somewhat diminished in amount and epithelial cells, leucocytes, and tube-casts, generally hyaline, are present, but seldom any blood. Dropsy is absent. Recovery is generally rapid. Nephritis is seen usually in the 1st or 2d week, and is, as a rule, much less dangerous and persistent than that connected with scarlet fever. In some severe instances a sudden, acute nephritis may develop, characterized by great diminution in the amount of urine, a high degree of albuminuria, and epithelial casts, yet only exceptionally by dropsy and symptoms of uremia. Chronic nephritis is an unusual sequel.

Diphtheritic Paralysis.—Observed as a complication during the attack diphtheritic neuritis manifests itself in the production of heart-failure, and occasionally, in severe cases, of an early palatal paralysis. Occurring in some form, as a complication or a sequel, it was seen in 20.7 per cent. of 2300 cases of diphtheria observed by Rolleston.³ As a sequel it is a cause of cardiac failure, but is especially seen in the "*postdiphtheritic paralysis*" affecting various regions of the body. It is observed oftenest after severe attacks in those past the period of infancy, but to this there are numerous exceptions, and not infrequently it occurs when the diphtheritic affection of the throat has been completely overlooked. The most frequent and usually the first seat is the soft palate, which exhibits paralysis most frequently 3 or 4 weeks or more after the onset of the attack of diphtheria. Baginsky⁴ found this symptom 68 times in 993 cases of diphtheria. The speech becomes nasal and fluids pass into the nose when attempts at swallowing are made. The palate is relaxed and does not move with phonation and the palatal reflex is abolished. With this paralysis is generally associated a loss of the patellar reflex, even in cases where no sign of neuritis is discoverable beyond the palate. Indeed the patellar reflex may be abolished without any palatal involvement. Early in the attack the paralysis may extend in some cases from the palate to the pharyngeal and laryngeal muscles, with the result that liquids readily enter the trachea when swallowing is attempted, and that respiration may be interfered with by the laryngeal involvement.

Next in frequency and in order of development, although much less common, is paralysis of the ocular muscles, producing strabismus, loss of accommodation, dilatation of the pupils and ptosis or sometimes other evidences of neuritis of the third nerve. The nerves of the face may exceptionally be attacked, and not uncommonly those of other parts of

¹ Boston Med. and Surg. Journ., 1904, CLI, 433.

² Nothnagel, Spec. Path. u. Therap., II, 226.

³ Arch. of Pediat., 1913, XXX, 335.

⁴ Loc. cit., 209.

the body, especially the lower extremities. In the latter event there develop paresthesia, pain, and difficulty in walking owing to lack of power or of coördination. The condition is that seen in multiple neuritis of any sort and the electrical reactions are the same. Less often the arms share in the ataxia and loss of power, and occasionally the head drops forward from involvement of the muscles supporting it. In bad cases the child may be almost powerless and respiration may become difficult through paralysis of the abdominal and thoracic muscles and of the diaphragm. Death may result from inability to swallow, interference with respiration, or the cardiac paralysis already referred to.

In the majority of cases, however, the paralysis affects only the palate and the patellar reflexes and often the ocular muscles as well, and recovery begins in about 2 weeks and advances rapidly. When the affection is more widespread, convalescence is much slower. Rarely there occurs a cerebral paralysis of the hemiplegic type. Dynkin¹ was able to collect 72 reported cases, and Rolleston² 80 cases.

Digestive disturbances are frequently seen in severe cases. The vomiting which attends heart-failure has already been referred to, Diarrhea may occur, dependent upon inflammation other than of a pseudomembraneous nature.

Prolonged anemia and debility are frequent sequels after severe cases and to some extent after all. Chronic *rhinitis* is a common sequel and *otitis media* is often seen, although less frequently than after other infectious diseases. It arises generally by infection through the Eustachian tube. Rolleston³ reported it in 4.10 per cent. of 5076 cases in the Metropolitan Asylums Board's Hospitals.

Various *cutaneous eruptions* may occur, among these being a diffuse multiform erythema. Herpes is sometimes seen (4 per cent. Rolleston).⁴ Purpuric eruptions may be observed in malignant cases. Various urticarial and erythematous eruptions develop after the use of antitoxin. (See Treatment.)

Other infectious diseases may be associated with diphtheria, prominent among these being scarlet fever and measles. Chicken-pox, small-pox, whooping cough and typhoid fever may occur in combination with diphtheria.

Among more unusual complications may be mentioned pleurisy, endocarditis, pericarditis, cutaneous emphysema—the result of necrosis in the pharynx—arthritis, meningitis, and thrombosis or embolism in the brain or extremities, the latter perhaps followed by gangrene. Ransome and Corner⁵ could collect but 9 cases of this from medical literature, including 1 of their own; and Rolleston⁶ but 11. I have seen it in 1 unreported instance, and another has been reported by Gunson.⁷

Relapse.—This is not as rare as often supposed, Rolleston⁸ finding it in about 1.5 per cent. of 2560 cases. It consists in the reappearance of the symptoms of the disease within a few weeks after the onset of the first attack and before the germs have left the system. The occurrence of measles during convalescence from diphtheria is liable to be followed by a re-development of the diphtheritic process.

¹ Jahrb. f. Kinderh., 1913, LXXVIII, Ergänzungsh., 267.

² Clin. Journ., 1913, XLII, 12.

³ Brit. Jour. Child. Dis., 1915, XII, 18.

⁴ Brit. Journ. Dermatol., 1907, XIX, 375.

⁵ Lancet, 1911, I, 94.

⁶ Brit. Jour. Child. Dis., 1910, VII, 529.

⁷ Brit. Jour. Child. Dis., 1916, XIII, 237.

⁸ Brit. Jour. Child. Dis., 1907, IV, 332.

Recurrence.—Recurrence is frequently seen, protection given by one attack appearing to continue but a very short time, probably only a few weeks or months, after which there is no certain immunity, and some individuals even seem predisposed to repeated attacks. Yet recurrences as compared with the total number of cases of diphtheria are not very common and are prone to be less severe than the first attack. It is uncertain to what extent this depends on the acquired immunity which increases as the individual grows older. (See p. 460.)

Prognosis.—The mortality of diphtheria, always high, has varied much in different periods, even before the introduction of antitoxin treatment. Epidemics have differed greatly in their severity, depending probably on varying degrees of virulence of the germ. Since the last portion of the 19th century the disease as a whole has certainly become less serious, although this diminishing severity has been affected in part by the employment of antitoxin, and in part by the application of bacteriological diagnosis, by which very many mild cases are recognized which would otherwise have passed undetected.

Yet the prognosis in individual cases is always most uncertain. Unfavorable symptoms may readily develop in cases which at first appeared to be mild, and unexpected complications and sequels may add very greatly to the danger. The favorable influence of the employment of antitoxin is certainly very great. This has been so uniform an experience that it no longer demands proof. A few statistics may, however, be given by way of illustration. Burrows¹ found that for 15 years prior to the introduction of antitoxin the mortality in Boston had been 30.8 per cent., whereas in 1962 cases treated in the Boston City Hospital in a single year after this period, the mortality had fallen to 12.23 per cent., or, if those moribund on admission were deducted, 9 per cent. The statistics of the New York Board of Health (Northrup)² showed a mortality of 34.9 per cent. in 27,210 cases treated without antitoxin, and only 15 per cent. in 56,425 cases treated with it. The report of the Metropolitan Asylum Board's Hospitals for London (Herringham)³ gave a mortality of 30.25 per cent. in 11,704 cases before the introduction of antitoxin, and from 22.5 to 9.29 per cent. in the years following this. Most of the published statistics are, to a large extent, from hospital practice, and are virtually alike in the diminished mortality shown. In purely private practice the results with antitoxin are still better. In 1610 such cases in St. Louis, collected by Zahorsky⁴ the mortality was but 1.5 per cent.

The favorable effects of antitoxin treatment have been especially marked in laryngeal cases. According to the investigations of the American Pediatric Society,⁵ before the employment of antitoxin about 73 per cent. of cases of laryngeal diphtheria died, while in 1704 cases treated with antitoxin in private practice the mortality was but 21.12 per cent. In 15,148 laryngeal cases occurring in the practice of New York City physicians, as quoted by Biggs and Guerard,⁶ all treated with antitoxin, the mortality was but 16.6 per cent. Moreover the employment of antitoxin certainly appears to have diminished greatly the necessity of operative treatment. The report of the American Pediatric Society showed that about 60 per cent. of the laryngeal cases thus treated did not

¹ *Loc. cit.*, 125.

² Nothnagel's Encyclopedia, American Ed., Diphtheria, 143.

³ Albutt and Rolleston's Syst. of Med., I, 1630.

⁴ Med. News, 1903, LXXXII, 1085.

⁵ Med. News, 1897, LXX, 632.

⁶ Med. News, 1896, LXIX, 677.

need intubation, while previously about 90 per cent. required it. The mortality also of cases requiring intubation has diminished under the influence of antitoxin. In 639 such cases without antitoxin reported by McCollom¹ there was a mortality of 82.49 per cent., while 1478 intubated cases receiving antitoxin had a mortality of only 41.4 per cent. Siegert's² 37,000 collected cases of tracheotomy and intubation showed a mortality of 60.55 per cent. before the employment of antitoxin and only 35.70 per cent. after this period.

The *promptness* with which the serum treatment is commenced is important. In the first report of the American Pediatric Society³ the mortality in cases injected on the 1st day of the disease equalled 4.9 per cent. This increased steadily to 22.9 per cent. in cases first treated on the 4th day and to 38.9 per cent. after the 4th day. Biggs and Guerard⁴ published the following table illustrative of this fact.

TABLE 67.—COMPARISON OF DATE OF TREATMENT AND MORTALITY

Date of treatment	Cases	Mortality, per cent.
1st day of the disease.....	1415	3.5
2d day of the disease.....	2640	8.0
3d day of the disease.....	2340	12.8
4th day of the disease.....	1458	23.6
5th day or later.....	1912	35.0

Various factors other than treatment influence the mortality. *Age* is prominent among these. The disease is much most fatal in infancy, largely on account of the danger of laryngeal involvement and the development of bronchopneumonia, and becomes progressively less dangerous as age advances. The great majority of deaths are in subjects less than 5 years old. In Burrows⁵ 1962 cases, all treated with antitoxin, the mortality was divided as follows: 1st year 40.40 per cent.; 2d year 33.9 per cent.; 3d year 23 per cent.; 4th year 15.60 per cent.; 5th year 14.60 per cent. The total mortality from birth to 5 years was 21.30 per cent., from 5 to 10 years 8.40 per cent., and from 10 to 15 years 3.10 per cent. In 17,889 fatal cases in the statistics of the New York Board of Health⁶ 14,554 (81.36 per cent.) were under 5 years of age. Favorable *social* and *hygienic conditions* improve the prognosis decidedly. The *situation of the membrane* and the rapidity of its extension influences the prognosis in individual cases. An abundant and rapidly spreading or deeply penetrating membrane in the throat makes the prognosis grave. Involvement of the larynx, as already stated, increases the mortality greatly. Primary nasal diphtheria is generally of little danger to the patient, although sometimes serious by its extension to the larynx. In diphtheria of the nasopharynx, on the other hand, the prognosis is unfavorable. The development of marked *septic symptoms* is an unfavorable prognostic indication as is also evidence of *cardiac weakness*. The Boston City Hospital Cases showed that the mortality was often directly in proportion to the rapidity of the pulse. The development of *hemor-*

¹ Royer, Proceedings Phila. County Med. Soc., 1905, XXVI, 80.

² Jahrbuch f. Kinderheilk., 1900, LII, 56.

³ Transac. Am. Ped. Soc., 1896, VIII, 21.

⁴ *Loc. cit.*, 728

⁵ *Loc. cit.*

⁶ Northrup, *Loc. cit.*, p. 24.

rhagic conditions makes the prognosis extremely serious. The absence of an increase of the polymorphonuclear cells in the blood has been considered of bad import, although this is not universally admitted.

Among serious complications are bronchopneumonia and an unusual degree of albuminuria, of anemia, or of lymphatic enlargement. Post-diphtheritic paralysis generally results favorably if the heart escapes. The combination of measles with diphtheria or the development of one after the other greatly increases the gravity of the case. Scarlet fever occurring as a complication is unfavorable and the case may be readily fatal. If the order is reversed, diphtheria being the complication, the prognosis is not so often affected. The association of diphtheria and typhoid fever is dangerous.

Diagnosis. Clinical.—Even without bacteriological examination the diagnosis of diphtheria is usually easy in typical and well-developed cases. Early in the disease or in atypical attacks it may be a matter of great difficulty. It rests, in general, in the case of tonsillar diphtheria upon the rapid development of pseudomembrane which is removed only with difficulty and which leaves a bleeding surface beneath; the reforming of the membrane after removal; its tendency to spread beyond the tonsils; constitutional depression out of proportion to the local symptoms; the frequent presence of albuminuria, and of glandular enlargement in the neck, and the common occurrence of paralysis as a sequel. The mode of onset and the temperature curve are too variable to be of much diagnostic assistance.

Mild attacks of diphtheria are most liable to be confounded with *follicular tonsillitis* especially when the separate foci of secretion in the latter disease fuse and cover the tonsil. The onset of this condition is generally more sudden, the fever higher; the throat feels sorer and is of a deeper red; the early swelling greater; the secretion is easily removed, and there is no spreading beyond the tonsils. The patient often has the history, too, of having been susceptible to repeated similar attacks. It is impossible, however, to make a positive diagnosis between the ordinary follicular tonsillitis and diphtheritic folliculitis without a bacteriological examination. Severe and even fatal *streptococcic inflammation of the tonsils* may occur, and can be distinguished from diphtheria only by bacteriological examination. Such cases are seen, for instance, in scarlet fever. Similarly a severe pseudomembranous inflammation is sometimes dependent upon *pneumococcic involvement*.

Primary nasal or nasopharyngeal diphtheria is often unrecognized because unsuspected. It is especially in infants and young children, when free nasal discharge is combined with excoriation of the lip and nostrils and perhaps the presence of fever, that the secretion should be studied bacteriologically and an examination for membrane made.

Laryngeal diphtheria must be distinguished from stenosis depending upon other laryngeal conditions. The persistence of stenosis by day as well as by night usually indicates the existence of something more than *false croup*, yet not necessarily so. The further differential diagnosis is considered under Respiratory Diseases. The severe laryngeal stenosis which sometimes accompanies measles may or may not be diphtheritic in nature. Only a laryngoscopic examination, when this can be made, or a bacteriological study can finally determine the question.

The diagnosis is sometimes difficult between diphtheria and *scarlet fever*, but only in anomalous cases. The former disease may exhibit a scarlatiniform erythema, while in the latter the eruption may be absent,

undiscovered, uncharacteristic, or late in appearing. The onset of scarlet fever, however, is usually more abrupt and the inflammation of the throat at the outset more severe. Only a bacteriological study can settle the diagnosis in some instances. The combination of the two diseases may be especially difficult to recognize. Here the sequence of symptoms is the best guide. *Thrush*, *aphthæ* and *ulcerative stomatitis* offer little practical difficulty in diagnosis. The first two are different in situation and appearance, and the last could suggest diphtheria only when this is limited to the oral cavity; an occurrence of great rarity. *Vincent's angina* may resemble diphtheria early in the attack, and a bacteriological examination may be required to distinguish them. The deposit in *Vincent's angina* is more liable to involve the tongue, cheeks, and gums. When on the tonsils it is usually confined to one side, is slower in developing, more necrotic than pseudomembranous, and tends to produce a deeper and more punched-out ulcer.

Bacteriological.—Although, therefore, the clinical diagnosis of diphtheria can be made with positiveness in many cases of the disease, the bacteriological diagnosis, as already indicated, is always of value and often indispensable. In all suspicious cases cultures should be taken as early as possible. The secretion is obtained by rubbing the affected area, or the posterior portion of the pharynx when the larynx is involved, firmly with a swab of sterilized cotton or a platinum wire loop and then transferring the germs in the same manner to the blood-serum culture-medium in a test-tube. After about 6 hours, or with more certainty after 12 hours, this may be examined for the presence of the diphtheria bacilli. The culture should not be taken immediately after an antiseptic application has been made to the suspected region. It can also not be depended upon if made late in the course of the disease. Since the surface of a thick membrane may fail to give a positive culture, inasmuch as the bacilli here are dead, a lower layer should be reached if possible. A single negative culture is not sufficient in suspicious cases. A test of the virulence of the germs is sometimes necessary, effected by injecting them into guinea-pigs. Sometimes the diagnosis of diphtheria can be made by the immediate staining of smear-preparations from the throat or nose. The failure to find the germs under these conditions is not, however, a proof that the disease is not diphtheria.

Regarding the diagnostic value of the diphtheria bacillus, while this is undoubtedly very great, reliance should not be placed on it alone. A case which is clinically diphtheria should be treated as such in spite of the failure to find the specific germ. On the other hand the mere presence of diphtheria bacilli in healthy throats does not constitute diphtheria (see p. 444) and there is no reason why the same accidental presence should not be found in persons whose throats exhibit a catarrhal or even a lacunar tonsillitis. Nevertheless, the combination of diphtheria bacilli with catarrhal pharyngitis or rhinitis generally indicates the existence of diphtheria and the provisional diagnosis of this disease should be made.

As to the diphtheroid bacilli other than the virulent Klebs-Löffler germ, there is much difference of opinion. There may be found in the throat an organism which is in reality the diphtheria bacillus although non-virulent to guinea-pigs. There is also described the pseudo-diphtheria bacillus of Hofmann-Wellenhof already referred to (p. 445). Neither germ is capable of producing diphtheria, and neither is common in cases which are apparently clinically this disease; but whether they are

in reality but variants of the diphtheria bacillus, capable under favorable circumstances of again becoming virulent, is not certainly determined. The difficulty in making a diagnosis of diphtheria solely upon the cultural findings is therefore great. Van Riemsdijk¹ found bacteria resembling diphtheria-bacilli in 50 per cent. of children examined in a region where there had been no diphtheria for 10 years, and Kolmer² reported diphtheria-bacilli, although avirulent, in 40 per cent. of cultures from the penis of 100 healthy boys.

Treatment. PROPHYLAXIS. Natural Immunity.—Bearing closely upon the necessity of immunization is that of natural immunity. It had been recognized that infants in the early months of life were less susceptible to diphtheria than after this period and during early childhood; and that through later childhood there was a slowly increasing return of immunity. The investigations of Schick³ have established these facts upon a scientific basis. He found that the intracutaneous injection of $\frac{1}{50}$ of the minimum dose of toxin lethal for a guinea-pig, diluted with 0.05 to 0.2 c.c. of normal salt solution, will produce in non-immune persons an erythema, while in those immune there is no reaction whatever. In making the test a small, accurately graduated syringe is employed, with a very fine special needle; the skin pinched into a fold; and the needle inserted into the *skin*, not beneath it. If no antitoxin is present in the patient's circulation, a red, slightly edematous spot of erythema, 0.5 to 2 cm. in diameter, with a brownish tinge, appears within 24 to 48 hours (Fig. 138). It begins to disappear within from 7 to 10 days, leaving a brownish pigmented area with superficial desquamation. The value of this test has been confirmed by other investigators, among them in the United States being Moody,⁴ Park, Zingher and Serota,⁵ Kolmer and Moshage,⁶ Shaw and Youland⁷ and others. A comparison of the statistics given by the writers mentioned, the figures being approximations only, shows that a positive reaction; *i.e.* an absence of immunity, was present in the new born in 7 per cent. (Schick); in the 1st year in 35 to 45 per cent.; from 1 to 2 years in 55 to 60 per cent.; from 2 to 5 years in about 66 per cent.; from 6 to 8 years in 35 to 45 per cent.; and from 8 to 15 years in 25 to 30 per cent. Tests by Zingher⁸ give decidedly lower figures. Pseudo-reactions are described as less sharply defined, more infiltrated, and disappearing in 48 hours.

The practical value of the Schick test is great, in that it enables us to determine what subjects are already immune. Those who give a negative reaction do not need antitoxin injections. This is a matter of moment from an economical point of view when considerable numbers of patients are to be immunized, and is important, too, when an anaphylactic reaction is feared in those who have previously received antitoxin. Since about 40 to 50 per cent. of children from 1 to 15 years are naturally immune (Kolmer and Moshage), it is evident that artificial immunization is required in only half of the children who have been exposed. In the first 6 months of life there appears to be in most cases a natural immunity.

¹ Niederl. Tijdsch. v. Geneesk., 1914, 1066.

² Arch. of Ped., 1912, XXIX, 94.

³ Münch. med. Woch., 1913, LX, 2608.

⁴ Journ. Amer. Med. Assoc., 1915, LXIV, 1206.

⁵ Arch. of Pediat., 1914, XXXI, 481.

⁶ Amer. Jour. Dis. Child., 1915, IX, 189.

⁷ Trans. Amer. Pediat. Soc., 1916, XXVIII, 329.

⁸ Amer. Jour. Dis. Child., 1916, XI, 269.

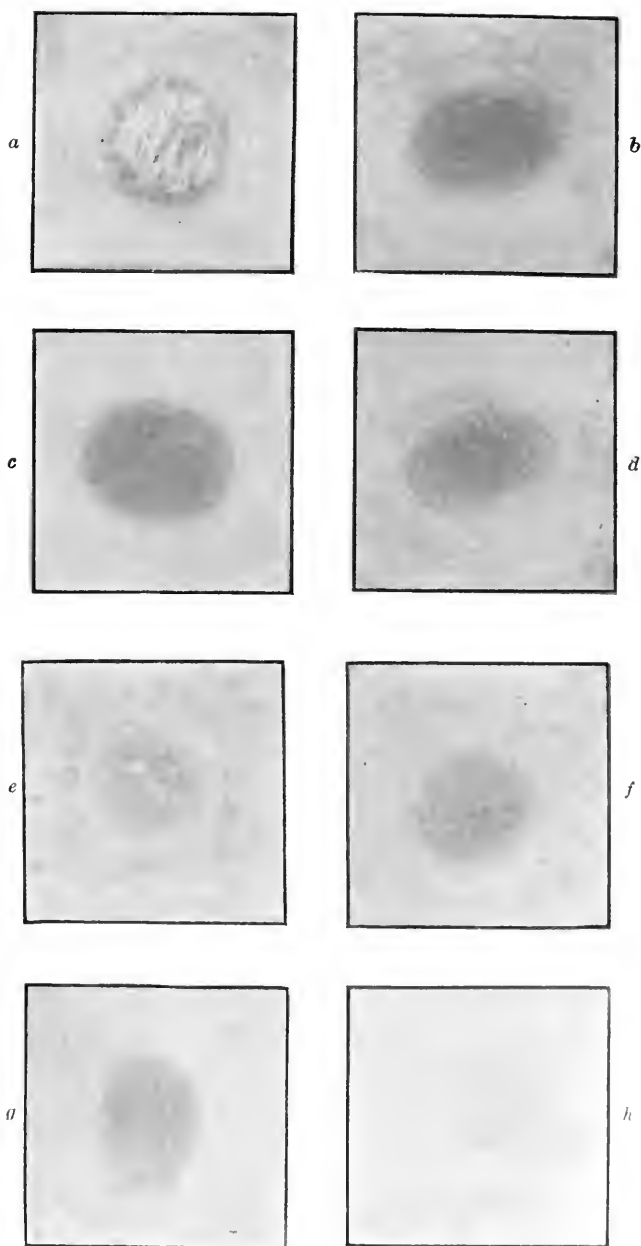
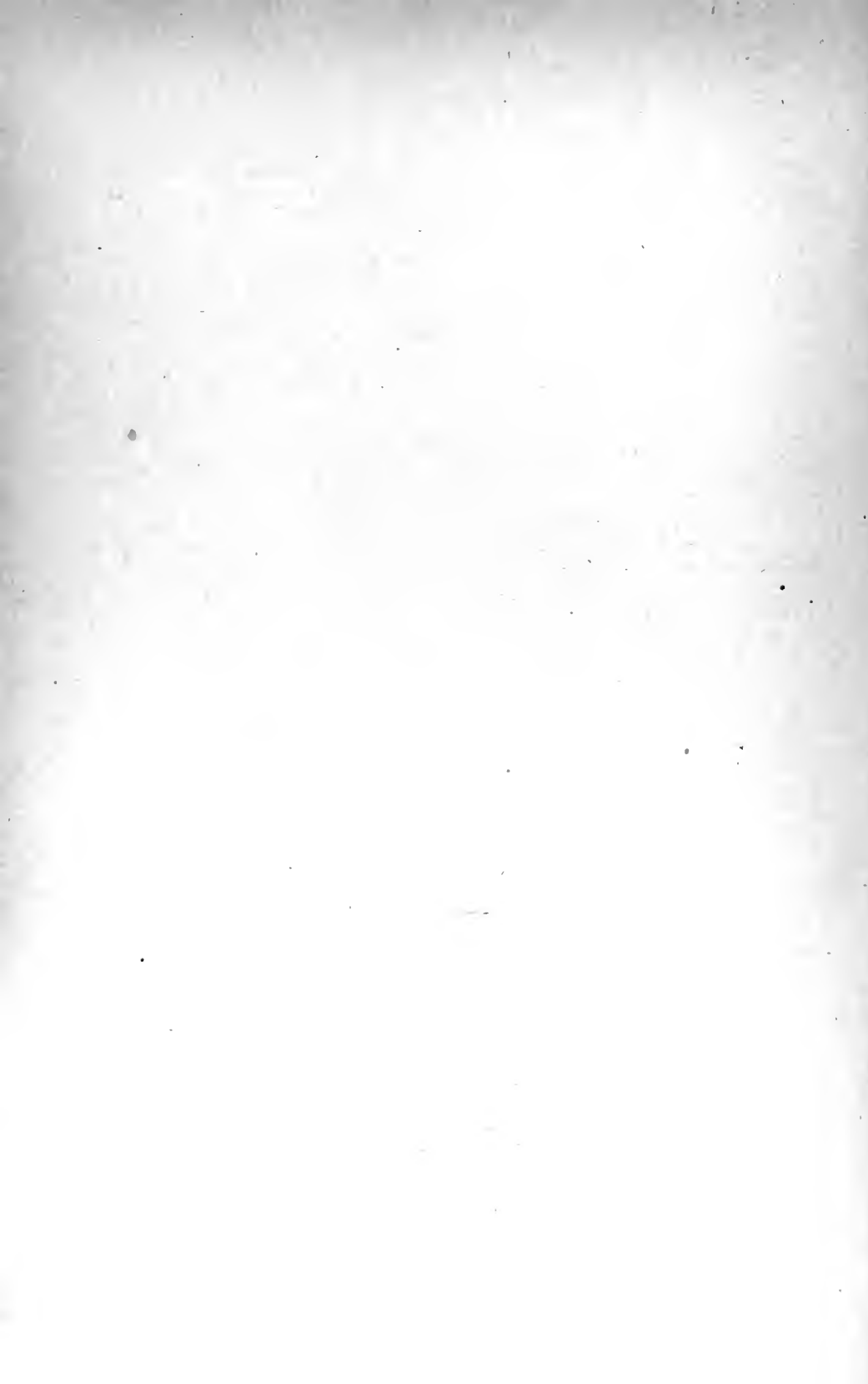


FIG. 138.—THE SCHICK REACTION IN DIPHTHERIA.

(a) to (d) Typical positive reactions 18 hours after test: (a) strongly positive reaction, with vesiculation of the surface layers of the epithelium, which is seen occasionally in individuals who have practically no antitoxin; (b) and (c) positive reactions; (d) a moderately positive reaction; (e) fading reaction 1 week after test; shows redness, scaling and beginning pigmentation; (f) 3, 4 or 2 weeks; (g) after 3 weeks; (h) faint pigmentation after 4 weeks. (Zapher, *American Journal of Diseases of Children*, April, 1916.)



Immunization.—Upon the development of a case of diphtheria, all susceptible members of the household, proven by the Schick test to be so, should receive an immunizing injection of diphtheria antitoxin. When for any reason the test cannot be carried out, certainly all the children of the family should be immunized, and it is no doubt safer to treat the older members in the same way. The dosage employed should be from 500 to 1000 units, the latter amount being used at all ages except the 1st year of life. The protection afforded is very complete yet but of short duration; not over 4 weeks and often not much longer than 10 days. Consequently children in hospital-wards should have the immunization repeated every 3 to 4 weeks, if a repetition of the Schick test gives a positive result.

In the effort to produce a more lasting immunity *v. Behring*¹ has urged repeated subcutaneous injections of a mixture of toxin and antitoxin, as first suggested for use in children by Theobald Smith in 1909.² The protective action is not effected until from 23 to 25 days (Schreiber),³ and often much later, but continues at least for months and perhaps for years. It is sometimes of advantage to combine with the toxin-antitoxin mixture a vaccine of dead diphtheria bacilli. Park and Zingher⁴ found that *v. Behring's* method of immunization greatly increased the amount of antitoxin in the blood of persons already immune; while in the case of those non-immune the treatment combined with the vaccine produces sooner or later an immunity in 40 out of 50 individuals treated. Inasmuch as the natural antitoxin is liable to be absent from the blood especially in infancy, Zingher⁵ advises that all infants below 18 months of age should be actively immunized, whether or not they exhibit a Schick reaction.

Quarantine.—Persons suffering from diphtheria should be isolated until at least 2 successive negative cultures show that the bacilli have disappeared from the mucous membranes. Persons merely suspected of having the disease ought to be quarantined until a bacteriological study can be made and the diagnosis determined positively. Other children in the family should be kept from school, and, if possible, removed from the house; and should not be allowed to mingle with others until bacteriological examination shows that no diphtheria bacilli are present on the mucous membranes. The nurse or members of the family in constant attendance upon the patient should employ disinfectant gargles and sprays, and should refrain from association with others. When cultures for any reason cannot be made—and this is now exceptional—the quarantine of the patient should continue for at least 3 weeks after the mucous membranes are free from a deposit. This ensures the disappearance of the bacilli in the very large majority of instances, since bacteriological examination shows that in probably 80 per cent. of cases there are none of these germs to be found in 1 week, and in 90 per cent. in 2 weeks, after the pseudomembrane has left the throat. In 882 cases recorded in the Department of Health of Philadelphia the average duration of quarantine from the onset of the disease to the 2d consecutive negative culture was 15.9 days (C. Y. White).⁶ Other details in connection with quarantine and disinfection are those appropriate to infectious diseases in general.

¹ Deut. med. Woch., 1913, XXXIX, 873.

² Journ. Exper. Med., 1909, XI, 241.

³ Deut. med. Woch., 1913, XXXIX, 928.

⁴ Bureau of Laboratories, City of New York, 1944-15, VIII, 104.

⁵ Amer. Jour. Dis. Child., 1918, XVI, 83.

⁶ Personal communication.

Management of Carriers.—The procedure for children of the family and for those in constant close contact with the patient has been referred to in the preceding paragraph. What is to be done with carriers of other sorts: *i.e.* those who without such known exposure are found to have diphtheria bacilli on the mucous membranes, is one of the most perplexing of problems. Theoretically it would be advisable to quarantine all such; but the studies of Pennington and others, already referred to (p. 445), showed the manifest impossibility of this procedure. Certainly when cases repeatedly develop in schools or hospitals, the only proper course is to make cultures from every individual, and to institute the proper treatment. (See Treatment of Carriers, p. 471.)

TREATMENT OF THE ATTACK. General and Hygienic.—The selection and the care of the sick-room are those described under the Management of Infectious Diseases (p. 306). Fresh air and sunlight are of great importance. The patient should be confined absolutely to bed in a recumbent position no matter how mild the attack. The diet should be liquid, preferably milk. The difficulty in swallowing and the distaste for food which sometimes develop later in the attack, often occasion such active resistance on the part of the child that the curtailing of the frequency of feeding may be necessary. This applies also to all local treatment in this disease, since exhaustion is such a prominent symptom. The question of the course to be pursued must be determined for each case individually. Feeding through a stomach tube or nasal tube is frequently a great aid in such cases.

The unusual prostration characteristic of diphtheria renders *stimulation*, especially alcoholic, necessary in all but the mildest cases. It is usually better to begin this early rather than to wait for visible evidences of exhaustion to manifest themselves. Comparatively large doses may be required, a child of 2 years seriously ill sometimes readily bearing from 1 to 2 fl. drams (4 to 8) of whiskey or its equivalent every 2 or 3 hours, the quantity depending upon the character of the pulse and of the heart-sounds and the general evidences of debility. In addition, digitalis, camphor, strychnine, adrenalin and nitroglycerine are frequently required to combat circulatory disturbances. These are often best given hypodermically. The internal administration of bichloride of mercury in doses of $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.001 to 0.002) every 2 hours was long in vogue in both pharyngeal and laryngeal diphtheria, and appears to have decided value. Tincture of the chloride of iron in full doses was for years a favorite remedy, but cannot be considered in any way a specific.

Serum Treatment.—Of the numerous remedies formerly in use little is now heard since the introduction and wonderful success of the *serum treatment*. (See Prognosis, p. 456, for statistics.) This method, first brought prominently before the medical profession by von Behring and Wernicke,¹ consists in the injection into the circulation of the patient of the serum of an animal—the horse being relied upon for this purpose—which has received repeated injections of the diphtheria toxin in increasing doses, and which, as a result, has developed in its blood a body, perhaps a globulin, powerfully antagonistic to the toxin, and hence called the anti-toxin. This neutralizes the toxin already in the patient's blood and checks the growth of the membrane by inhibiting further development of the bacilli. The strength and dosage of the serum is measured by "anti-

¹ Zeitschr. f. Hyg., 1892, XII, 10.



FIG. 243.—A HARD PROTEIN CURD, BROKEN INTO TWO PORTIONS.



FIG. 244.—THE SOAP STOOL.
Shows the white, salve-like character.



FIG. 245.—THE CURDY STOOL.

Shows the white, fatty masses, with mucus of a pale-brownish tint.

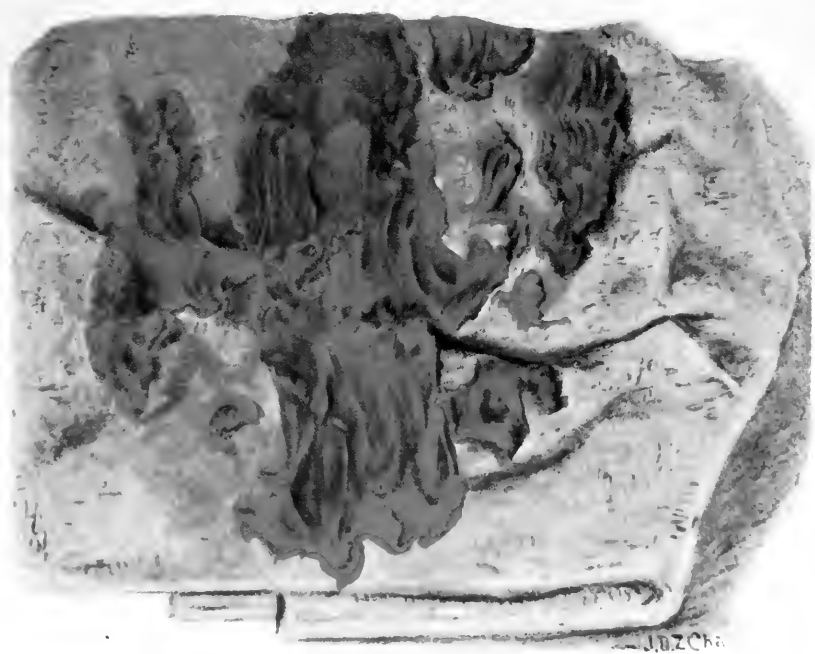


FIG. 246.—THE CARBOHYDRATE STOOL.
Smooth, soft, homogeneous, brown mass. Infant fed on malt-soup.

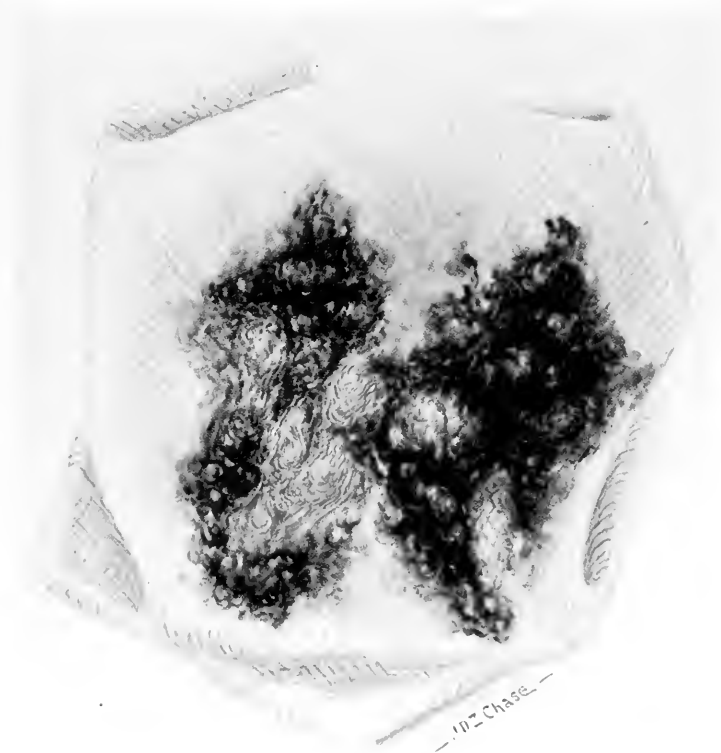
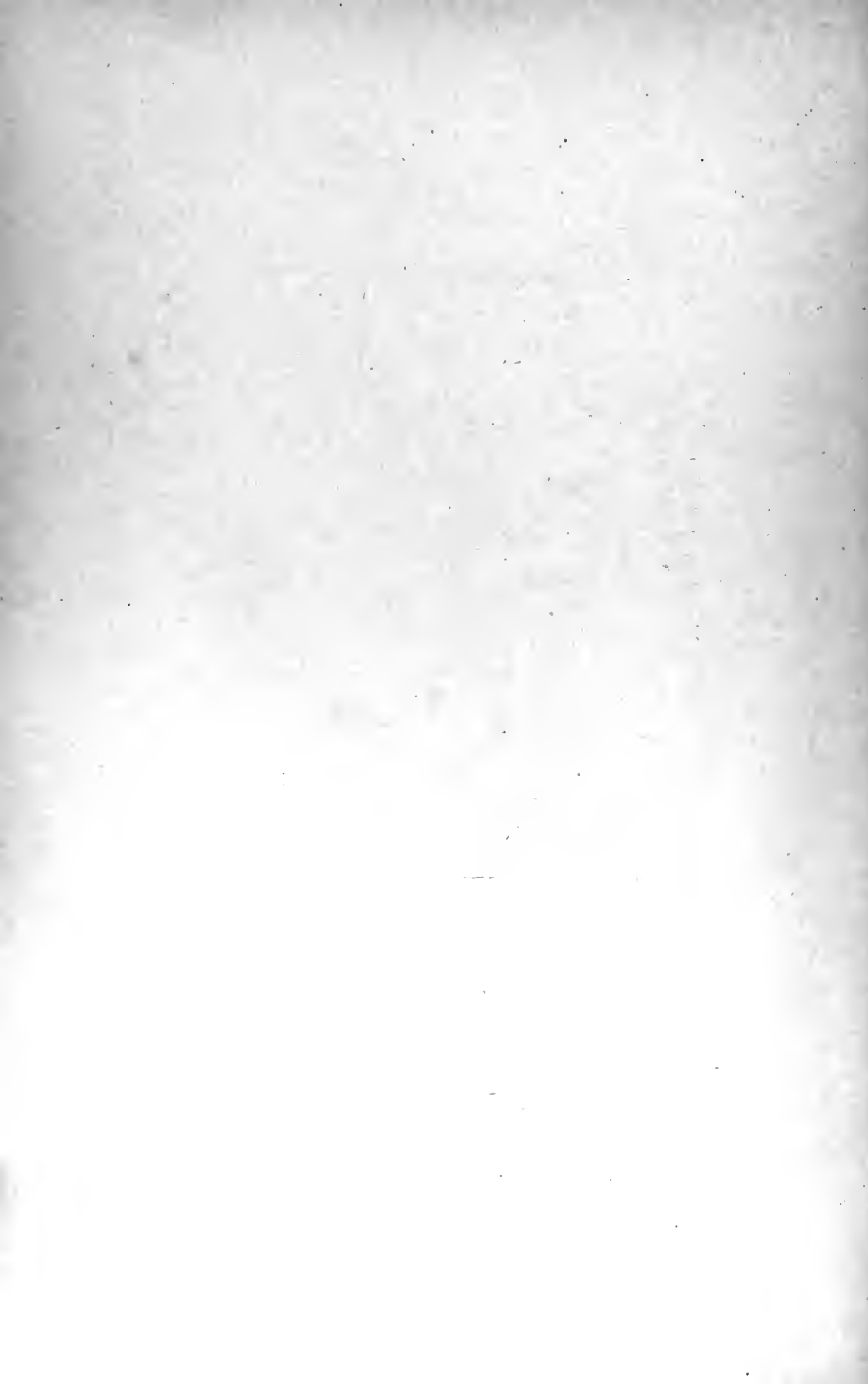


FIG. 247.—THE SPINACH-GREEN STOOL.

With a few lumps of fat-curd and large amount of mucus, a portion of it blood-stained.



toxin units," such a unit being the amount of serum sufficient to neutralize the effect of 100 times the dose of diphtheria toxin which would kill in 4 days a guinea-pig weighing 250 grams (8.82 oz.). The antitoxin serum is preserved in hermetically sealed glass vessels. It should be used promptly after it is opened.

Method of Employment.—The antitoxin can be given with a special syringe made for the purpose and thoroughly sterilized just before using. Makers now frequently supply the serum in a glass tube, which can itself be used as a syringe, a glass piston and hypodermic needles already sterilized accompanying this. A region should be chosen which is not pressed upon in lying, the flank and the abdomen being favorable situations. The skin should be thoroughly scrubbed with diluted alcohol and the hands of the operator cleansed in like manner. The air should be expelled before the needle is thrust under the skin. Local anesthesia is not necessary. A small piece of adhesive plaster or a pledget of cotton afterward painted with collodion may be applied after the needle is withdrawn.

Dosage.—For immunizing purposes the amount to be given varies from 500 units in the 1st year of life up to 1000 units for older children. In average cases of the disease over 2 years of age seen early 3000 to 5000 units should be given. If no improvement follows, this may be repeated within 6 to 12 hours and the treatment continued perhaps in larger doses until improvement is seen. In all severer cases in which the deposit has extended beyond the tonsils, and in all instances where involvement of the larynx is threatened, the initial dose should be 7000 to 10,000 units, varying with the age, and best given intravenously. These doses are larger than formerly recommended, but it is better to give more than really needed than to use too little, since the effects are practically never harmful.

Time of Administration.—The earlier in the disease the antitoxin is administered the more certain and powerful the action and the smaller the amount required to be effective. This is due to the fact that the antitoxin stops the destructive action of the diphtheria toxin, but cannot regenerate the cells which have already been injured by it. The comparative results of treatment begun on different days is very strikingly shown in the statistics detailed under Prognosis (p. 457). When given after the 3d day of the disease much less good can be expected. Nevertheless, as there are exceptions to the rule, no case should be considered to have lasted too long to seek benefit from serum treatment, and the dose should be large; while on the other hand very severe cases may be already too far advanced, even upon the 1st day, to be capable of being benefited. Even in the mildest and the doubtful cases, by far the wisest course is to administer antitoxin immediately without waiting for confirmation of the diagnosis by bacteriological examination.

Results of Antitoxin Treatment.—Favorable results following the injection are seen within 12 to 24 hours and sometimes earlier. They consist in a softening and separating of the pseudomembrane at its edges, a diminution of the glandular swelling and of nasal discharge, and an improvement in the general condition. In many cases the membrane disintegrates and disappears with surprising rapidity and the throat is clear in from 3 to 4 days. If no effects are seen the administration of the serum is repeated as described. Complications of diphtheria depending on damage already done by the toxin or upon septic conditions are in no way benefited by antitoxin. Thus the serum treatment does not appear

to diminish the number of cases of paralysis, since the damage done to the cells of the nervous tissue takes place very early in the attack. In favorable laryngeal cases there is a rapid lessening of the stenosis. The good effects of antitoxin are particularly evident in the reduction of the number of tracheotomies and intubations now necessary, as well as in the mortality where operation has been required. (See Prognosis, p. 456.)

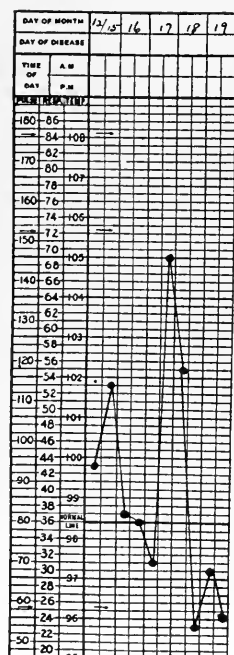


FIG. 139.

FIG. 139.—RISE OF TEMPERATURE PROMPTLY AFTER PROPHYLACTIC ADMINISTRATION OF DIPHTHERIA ANTITOXIN.

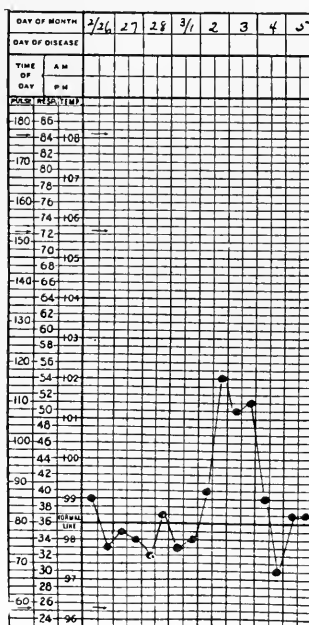


FIG. 140.

FIG. 140.—FEBRILE REACTION PROMPTLY DEVELOPING AFTER DIPHTHERIA ANTITOXIN.

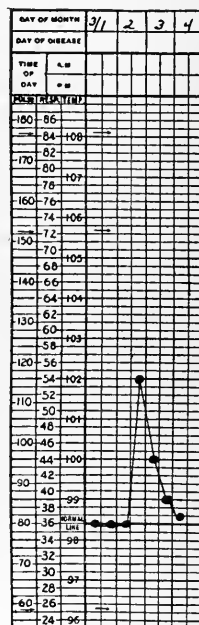


FIG. 141.

FIG. 141.—SERUM SICKNESS OCCURRING 7 DAYS AFTER THE ADMINISTRATION OF ANTITOXIN.

Dorothy McC., 7 years old. Immunizing dose of diphtheria antitoxin (2500 units) given on Feb. 23; throat being slightly red and cultures positive; Feb. 25, cultures being still positive, antitoxin repeated; Mar. 2, widespread eruption of urticaria which is giving pain; Mar. 3, still widespread typical antitoxin rash; Mar. 4, rash about gone.

Unfavorable Effects of Antitoxin.—A group of symptoms to which the title "Serum Disease" has been applied, not infrequently follows the administration of antitoxic serum. As this condition is due to the introduction of a foreign blood-serum independently of its content of diphtheria-antitoxin contained, it would seem that the smaller the amount of serum used the better. Consequently concentrated sera are always to be preferred as less liable to produce unpleasant after-effects and less painful at the time of injection. It is not, however, certain that the quantity of serum used bears any real relationship to the pro-

duction of symptoms. The symptoms are seen in from 5 to 30 per cent. of the cases, and occur anywhere from the 1st to the 17th day, although oftenest from the 5th to the 9th (Sturtevant).¹ They consist in erythema of a scarlatiniform or rubeloid type, urticaria, edema, fever (Figs. 139, 140, 141), vomiting, painful articular swelling, albuminuria, malaise, headache, and general aching. Some or all of these may be present, urticaria, often widespread and lasting 2 to 3 days, being the most frequent. (See p. 435, Fig. 127.) Instances of sudden death following the injection have occurred, but these have appeared, certainly in some cases, to be due to the mere hypodermic puncture in cases of patients suffering from lymphatism. (See Sudden Death, p. 216.) In other cases grave symptoms or death have apparently been due to an unusual susceptibility to the action of an alien serum, such as that of the horse. It does not appear proven that the result is produced by the antitoxin itself. The symptoms in these cases consist of alarming dyspnea and cyanosis developing promptly after the injection, and either passing away gradually or terminating fatally in a few minutes. It is true that repeated injections of antitoxin at intervals separated by months or longer may produce an increasing sensitiveness to the serum, but the majority of cases of sudden death have taken place after the period of childhood, after the first injection, and in individuals with a decided asthmatic tendency, especially produced by approach to a horse. Whatever the cause, the numbers of instances of dangerous symptoms or fatal result are so extremely few, as compared with the frequency with which antitoxin has been employed, that they must be ignored for practical purposes. As the serum has no action, either good or bad, upon complications, it should be given regardless of their presence. In persons with a positive asthmatic history it may be well to omit the administering of immunizing doses, or, in those with the disease, to defer it if the attack is mild. In those positively requiring the use of serum it should be given in a minute injection of a few drops at a time only, with pauses of a few minutes between, in order to observe the development of symptoms, if any are to appear.

Local Treatment.—Local treatment is of value in so far as it destroys or removes the bacilli from the affected mucous membrane, or retards their growth or checks their dissemination by producing conditions of cleanliness. It is harmful when it exhausts the patient; and inasmuch as exhaustion occurs so readily in this disease local treatment must be employed with great discrimination. The chief object is that of cleansing. For this purpose douching of the nose and throat with warm alkaline antiseptic solutions, such as a diluted liquor sodii boratis comp. (Dobell's solution) may be employed, or a normal salt or a 1 or 2 per cent. boric acid solution. The injection is preferably given with a fountain-syringe or a soft rubber nasal syringe (p. 238, Fig. 38). The child should lie well on one side and the fluid, if used in the nose, be made to enter the upper nostril and flow from the other. In some cases an atomizer may well be employed, but the syringe is generally more effective. Local treatment every 3 or 4 hours is enough in mild cases, and in severe ones every 2 hours. Spraying or swabbing the throat with a much diluted and non-acid solution of peroxide of hydrogen is often efficacious in dissolving the membrane. Application of solutions of more powerful drugs, such

¹ Arch. Int. Med., 1916, XVII, 176.

as preparations of iron, nitrate of silver, and the like, have largely gone out of vogue. They are usually painful and cause distress to and resistance by the patient. An ice-bag applied externally over the position of the tonsils, and small pieces of ice placed frequently in the mouth, tend to relieve the pain and difficulty in swallowing.

Extension of the disease to the larynx is largely prevented by the early and free use of antitoxin. When, however, laryngeal symptoms are threatening, the use of the croup-tent (p. 236) is often of great benefit. The patient should, however, not be kept in the tent over 20 or 30 minutes at one time, as the effect may be depressing or the amount of oxygen too greatly diminished. Sometimes allowing a stream of oxygen to enter the tent at the same time with the vapor is of benefit.

Operative Treatment.—When cyanosis is clearly increasing in laryngeal cases, stenosis is decided, and the accessory muscles of respiration in full play, prompt operative measures are indicated, and it is important that they be not unduly delayed. These consist respectively in *tracheotomy* and in *intubation*. The latter has largely supplanted the former in most countries. Tracheotomy, however, may quickly become necessary after intubation if no relief is obtained.

The technique of *tracheotomy* is so strictly surgical that no attempt will be made to describe it in this connection. The after-treatment of tracheotomy consists principally in keeping the tube clean of mucus and pseudomembrane, and in maintaining the air saturated with warm water-vapor produced by the boiling of water, the slaking of lime, the plunging of hot iron into water, or in other ways.

Intubation.—This was first successfully put into practice by O'Dwyer¹ and has been the means of saving many lives. Inasmuch as favorable results depend to a large extent on the skill and training of the operator, I append the following description kindly prepared for me by Dr. Henry R. Wharton, Senior Surgeon to the Children's Hospital of Philadelphia.

"Instruments Required for Intubation.—The instruments required for intubation are *intubation tubes*, and an apparatus to insert them. The tubes for children are usually six in number, of different sizes adapted to the age of from 1 to 12 years. The sort now generally employed consists of a metallic cylinder which bulges near its center, with a collar or head to rest upon the vocal cords. The tubes are gold plated or of hard rubber with a metallic lining, and are provided with an obturator which has a blunt extremity; and through the edge of the collar on each tube there is a small perforation into which a strand of fine braided silk is passed. This serves to remove the tube, if in its introduction it should have been passed into the pharynx or the esophagus instead of the larynx, or if it has to be hurriedly withdrawn, owing to sudden obstruction of breathing.

"The Introducer.—This instrument consists of a handle and a staff, bent to a right angle at its extremity, having a screw that attaches it to the obturator, and a sliding gear for detaching the obturator from the tube when it is placed in the larynx (Fig. 142).

"Mouth-gags.—Mouth-gags of various kinds may be employed. The one generally used is a self-retaining instrument (Fig. 142), and the portion inserted between the teeth is covered by pieces of rubber-tubing to prevent injury.

"Extractor.—The extractor is also bent at a right angle and has at its extremity a small forceps with duck-bill blades which are made to sepa-

¹ N. Y. Med. Journ., 1885, XLII, 145.

rate and apply themselves to the inner surfaces of the tube with sufficient firmness to withdraw it (Fig. 142).

Preparations for Intubation.—It should not be forgotten that when an intubation tube enters the larynx breathing is arrested until the obturator is removed, and therefore the manipulations should be as rapid as is consistent with accuracy. The surgeon should select a tube of suitable size for the age of the patient, pass a strand of fine braided silk about 2 feet in length through the opening in the collar of the tube, and knot the ends together. The tube is then attached by means of the obturator to the introducer. To prevent the patient from biting the finger, in case the mouth-gag should slip, the surgeon should protect the index-finger of



FIG. 142.—O'DWYER'S INTUBATION INSTRUMENTS.

A, Tube with obturator; B, tube; C, obturator; D, metal gag; E, mouth-gag; F, introducer; G, extractor; H, silk cord. (Fowler.)

the left hand in the region of the second joint by wrapping it with a piece of rubber plaster, or by slipping over it a metal shield.

Operation.—This may be performed while the child is in the sitting posture, or while it is recumbent. The former position I prefer. The child should be placed upon the lap of the nurse or assistant, and covered by a blanket loosely thrown around it. The nurse grasps the child's elbows from outside of the blanket and holds them firmly, but should not press them against the chest in such a way as to embarrass the respiratory movements. At the same time the legs of the patient should be secured by being held between the knees of the nurse. The head of the patient is held firmly between the open hands of the assistant, placed on either side of the head and cheeks. The left hand of the assistant may also be used to steady the mouth-gag after it has been introduced (Fig. 143).

As before stated, the tube may be introduced with the child in the recumbent posture. This I have done when, from the condition of the circulation, I did not think it advisable to lift the patient to the sitting posture. The mouth is opened and the blades of the mouth-gag introduced between the molar teeth upon the left side, and the jaws opened by this as widely as possible. The surgeon next passes the index-finger of the left hand into



FIG. 143.—INTUBATION. INSERTING THE TUBE.

(Wharton, *Starr's Amer. Text-book of Diseases of Children*, 1894, 314.)

the pharynx and feels for the epiglottis, hooking this forward by the end of the finger. The tube attached to the introducer held in the right hand is next passed into the mouth and carried back to the pharynx, the operator being careful to see that it hugs the base of the tongue in the middle line, that the handle is depressed well upon the child's chest, and that the silken loop is free. When the extremity of the tube comes in contact with the end of the finger resting upon the epiglottis, the handle of the instrument should be raised as the tube enters into the larynx and descends into

that organ, and when in position, the finger is placed upon the head of the tube to prevent its being withdrawn with the obturator. The trigger is next pressed, and the introducer and obturator withdrawn from the mouth by depressing the handle upon the chest. Before removing the finger it is well to push the tube well into the larynx. As soon as the obturator is removed there is generally a violent expiratory effort with coughing, accompanied by a gush of mucopurulent matter or membrane, and after this escapes the breathing is usually satisfactorily established. If the operator has passed the tube into the pharynx or esophagus, no improvement in the respiration takes place, and it should then be withdrawn by the silken loop and another attempt made to introduce it correctly. The mistake which inexperienced operators make in attempting to introduce the intubation tube consists in not hugging the posterior surface of the tongue closely, as a result of which the tube passes over the epiglottis into the pharynx. The most serious complication which is apt to occur during the introduction is in the pushing of a mass of membrane in front of the tube into the trachea. If this is too large to be expelled through the tube, the breathing is suddenly arrested. The tube should then be removed at once, and if the mass of membrane does not escape upon the expiratory efforts of the patient, the trachea should be rapidly opened. So much do I dread this accident, which has occurred to me in one case only, that I never introduce an intubation tube without having at hand the necessary instruments for a rapid tracheotomy.

"Another accident which is said to have occurred, of which I have no personal experience, is the pushing of the intubation tube through the wall of the larynx into the cellular tissue. This is not likely to happen unless undue force has been used. The production of a false passage is recognized by the fact that, although the tip of the tube can be felt to enter the larynx, it does not descend, but projects above the epiglottis. Some operators keep the silken loop attached to the tube during the time it is retained in the larynx, so that by drawing upon it the nurse or attendant is able to withdraw the tube instantly if it should become obstructed with membrane, or be coughed up and pass into the pharynx or esophagus. I generally allow the loop to remain in place for 10 or 15 minutes. At the end of this time I introduce the finger into the mouth and feel that the tube is in its proper place, and while the tip of the finger rests upon the edge of the tube, divide the silk loop and withdraw it.

"*After-Treatment.*—After intubation, so far as the tube itself is concerned, no treatment is required. The patient should be kept in a warm room in which a certain amount of moisture is maintained by the use of boiling water or by a steam spray. If there is but little tendency to expectoration through the tube, soda solution, which consists of carbonate of soda, 1 to 2 dr. (3.9 to 7.8); glycerine, 1 fl.oz. (30); and water, 6 fl.oz. (177) applied by means of a steam atomizer, may be used with advantage. One of the greatest troubles after intubation of the larynx is the satisfactory feeding of the patient and the administration of liquid medicines. Liquids, as a rule, are not swallowed well, a portion of them passing into the tube and producing violent coughing. Cases are, however, occasionally met with in which the swallowing of liquids does not seem to be specially interfered with by the presence of the intubation tube. Nursing infants may sometimes continue at the breast after the operation. I usually order a diet of semisolids, such as corn-starch, soft boiled eggs, mush, and junket. The taking of a sufficient quantity of water often causes trouble, and in such cases the child may be allowed to swallow small pieces of ice, or

water may be regularly administered by the rectum. In cases where there is difficulty in swallowing even this form of diet, it may be necessary to resort to introduction of liquids into the stomach by means of a feeding-tube passed through the nostril into the esophagus. In young patients in whom a liquid or milk diet is essential, if the head is dropped a little

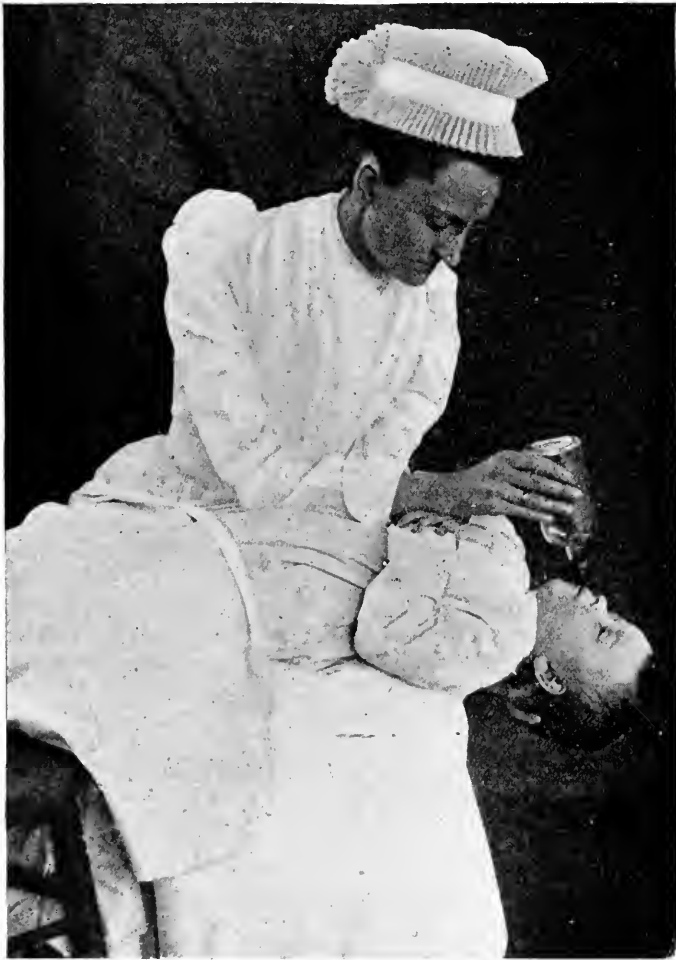


FIG. 144.—METHOD OF FEEDING INFANT AFTER INTUBATION, WITH THE HEAD LOWER THAN THE BODY.

(Wharton, *Starr's Amer. Text-book of Diseases of Children*, 1894, 316.)

lower than the body during the act of deglutition it will often be found that fluids are swallowed without difficulty (Fig. 144).

Removal of Intubation Tubes.—The intubation tube usually remains in place about a week. I usually remove it within 3 or 4 days, and if the breathing is satisfactorily carried on for half an hour, and no dyspnea appears, its reintroduction may not be necessary. If, however, after it has been out a few minutes, dyspnea returns, it should be promptly

reintroduced and its removal should not be attempted for 3 or 4 days. In many cases the tube is coughed out within a week from its introduction, and its reintroduction is not often required in these cases. It can usually be permanently dispensed with in from 5 to 10 days, although I have had cases in which it could not be permanently removed until the 15th day. Cases have been reported in which it had to be worn for many months. After an intubation tube had been coughed up or removed, the patient should be carefully watched from 12 to 24 hours, for the dyspnea may return at any time within this period and require replacement of the tube. The intubation tube may be coughed up and swallowed, entering the stomach. This accident need cause no anxiety, as in my experience these tubes usually pass safely through the intestines. After intubation of the larynx very decided hoarseness often persists for several weeks, but after this time usually entirely passes away.

"Retained Intubation Tubes.—If an intubation tube has been worn for a long time there is sometimes great difficulty in removing it permanently. Its removal may often be accomplished by introducing at intervals tubes of gradually increasing size."

Treatment of Complications and Sequels and of the Convalescence.—The exhaustion and *cardiac weakness* following severe cases of diphtheria require especial attention. Rest in bed in the recumbent position must continue so long as there are any evidences of decided weakness of the heart, shown by rapidity, irregularity, or slowness of the pulse, or by weak heart-sounds. Excitement and physical exertion of all sorts must be carefully avoided. Even after mild attacks the patient should be kept in bed for at least a week after the membrane has disappeared and only cautiously allowed to sit upright, although the heart may appear to be entirely normal. General tonic treatment is indicated, especially with alcohol, strychnine and sometimes digitalis. Adrenalin chloride (1:1000) has been used to prevent cardiac failure, the dose being 5 to 10 m. (0.31 to 0.616) according to age. Pituitrin has also been used with success.

The *anemia* which often remains demands a long course of iron or arsenic with abundant nourishment. Frequently cod-liver oil is of benefit, and the continued use of alcoholic stimulants may be needed. *Nephritis* remaining as a sequel requires treatment appropriate to it. It may necessitate a continuance of liquid diet, especially milk, longer than one would wish in view of the importance of nourishment from other points of view.

The treatment of *post-diphtheritic paralysis* depends to some extent upon the part affected. Paralysis of the extremities is to be aided by massage. That of the muscles of deglutition may occasionally require feeding by gavage, but this is uncommon. In any form of paralysis the chief aid is to be sought in electricity and the free use of strychnine combined with abundant nourishment.

Treatment of Carriers.—Finally, there is often difficulty in ridding the patient of the Klebs-Löffler bacilli which may persist long after all symptoms have disappeared, the individual thus becoming a carrier. The treatment is the same for healthy individuals who have not had the disease, but who have been proven to be dangerous carriers. The difficulty in getting rid of the bacilli in these cases is often very great. This is doubtless because in many instances they may remain indefinitely in the crypts of the tonsils or adenoid growths or in the nasal sinuses. Various measures have been recommended. A very weak (1:10,000)

solution of bichloride of mercury may be employed by syringing the nose or as a gargle; or a weak solution of boric acid or of liquor sodii boratis comp. may be used. Hand¹ applied with success a strong solution of nitrate of silver (60 gr.: 1 fl.oz.) (4:30) to the throat. Many report success with the spraying of the nose and throat several times daily with a boullion-culture of the staphylococcus pyogenes aureus, as first recommended by Schiötz.² A rapid disappearance of the diphtheria-bacilli may occur, these being crowded out by the other germs. (See articles by Lorenz and Ravenel,³ Rolleston,⁴ Lake⁵ and others.) Good results have also been claimed from the application of cultures of lactic acid bacilli applied in a similar manner (Nicholson and Hogan).⁶ Vaccine treatment with killed diphtheria bacilli has also been recommended, but the results as reported by Park and Zingher⁷ have been disappointing. In every case of persistence of the bacilli an inoculation test in guinea-pigs should be made. If the germs are proven to be non-virulent, quarantine is no longer necessary. In other cases of unusually long persistence of virulent bacilli, often the best means of treatment is the removal of the tonsils and adenoids.

CHAPTER XIII

GRIPPE

(Influenza)

Grippe, or influenza, was described clearly in the 12th century, and its epidemic, infectious character recognized since the 16th century. It first appeared in the United States in 1627. Whether the cases observed in these earlier widespread epidemics were etiologically identical in nature with those which have been encountered in more recent years cannot be positively determined; but from a clinical point of view no sharp distinction can be drawn. From a bacteriological standpoint a distinction may, it is true, be made, and it is to those depending upon the influenza bacillus that the title "influenza vera" has been applied, others resembling these clinically but being produced by other germs having been called pseudo-influenza, or "influenza nostras." It has also been proposed to designate as influenza all the diseases depending upon the influenza bacillus; a course very similar to that resulting should we call "pneumonia" all lesions produced by the pneumococcus. It seems best, therefore, to class together all those cases showing symptoms which have usually been regarded as evidences of grippe or of influenza respectively, and in which no sharp clinical distinction can be made, and to designate these "grippe," whatever the active germ may be. Certainly the mere existence of symptoms of a severe febrile cold does not warrant the diagnosis of grippe. Certainly, too, the epidemic contagious condition to which the title "grippe" is applied is as often dependent upon other germs

¹ Phila. Med. Journ., 1898, Aug. 24.

² Ugeskr. f. Læger, 1909, LXXI, No. 49. Ref. Journ. Amer. Med. Assoc., 1910, LIV, 442.

³ Journ. Amer. Med. Assoc., 1912, LIX, 690.

⁴ Brit. Journ. Child. Dis., 1913, X, 298.

⁵ New York Med. Rec., 1912, LXXXI, 1228.

⁶ Journ. Amer. Med. Assoc., 1914, LXII, 510.

⁷ Loc. cit.

as upon the influenza bacillus. The subject of grippe in early life has been exhaustively studied by Risel¹ with extensive bibliography.

Etiology. Predisposing Causes.—Climate, race, locality, sex, and social conditions, exert no influence. The previous health is also not a factor, except that affections of the respiratory tract increase the susceptibility. Consequently all causes are important which render the mucous membrane of the respiratory tract sensitive, such as bad weather, exposure to cold, insufficient ventilation, and the like.

All periods of life are susceptible, and the disease may occur even in the new born. Strassman² observed 8 cases at this period. In Comby's statistics³ of 218 cases in children, 48 occurred from birth to 2 years, 1 being but 17 days old; 76 at from 2 to 5 years, and 94 at from 5 to 15 years. The *epidemic character* is more marked than perhaps in any other affection. The disease at first occurred in epidemics, generally wide-spread and separated by decades. The great epidemic of 1889 appeared first in Turkestan after an interval of many years, and extended rapidly over the greater part of the earth. In less than 6 months it had reached the United States. In 1890-91 there was another serious outbreak most marked in England and America. The disease then became endemic to a limited extent, with occasional larger and more wide-spread outbreaks, at first regardless of season, but later usually in the cooler months of the year; until the greatest and most serious epidemic of it ever experienced, which occurred in 1918. This apparently arose in the Orient⁴ and spread rapidly over Europe and America, millions of individuals being attacked. Of the cities of the United States, Philadelphia suffered very heavily, about 150,000 cases having occurred in the course of 2½ months.⁵ The *individual susceptibility* is extreme, the large majority of persons exposed contracting the disease. This was especially true in the epidemic of 1889-90 and of 1918. In the last epidemic children were somewhat less frequently and decidedly less severely attacked than adults.

Exciting Cause.—The disease is a distinctly infectious one. The investigations of Pfeiffer⁶ revealed an extremely small, non-motile bacillus in the sputum and the nasal secretion, at first free in the mucus and later in the pus cells. It is less often present in the blood and it has been recovered from the cerebrospinal fluid by lumbar puncture. It generally disappears with the return of health, and it lives outside of the body in infected nasal or bronchial mucus for only about 14 days. Exceptions occur, however, and in more chronic cases or even in those entirely convalescent it can sometimes be found in the secretions or in the pus of complicating conditions even for months. According to Lord⁷ it could be discovered in the sputum years after recovery from the disease. Although the influenza bacillus appears to have been that most frequently present in the early epidemics, as time passed it was more and more replaced by other germs, and up to the year 1918 this bacillus could not be found in the large majority of cases. In the 1918 epidemic the opinions regarding the exciting cause were most divergent; in some regions the influenza bacillus being reported in the majority of cases; in others

¹ Ergebn. der inn. Med. u. Kinderh., 1912, VIII, 211.

² Zeitschr. f. Geburtsh. u. Gyn., 1890, XIX, 39.

³ Bull. de la soc. des hôp., 1890, VII, 67.

⁴ U. S. Publ. Health Service, 1918, Suppl. No. 34, Sept. 28.

⁵ Monthly Bulletin, Dept. of Health, 1918, III, No. 10-11, 23.

⁶ Deut. med. Woch., 1892, XVIII, 28; Zeitschr. f. Hyg., 1893, XI, 357.

⁷ Bost. Med. and Surg. Journ., 1905, CLII, 537.

its occurrence being comparatively uncommon and other organisms of various sorts being apparently the agents. Among the other germs recorded are the micrococcus catarrhalis, pneumococcus, staphylococcus, bacillus mucosus capsulatus, streptococcus mucosus capsulatus, streptococcus hæmolyticus, diplococcus mucosus and pneumococcus mucosus. These germs may occur in practically pure culture or often associated in various ways. Yet it is uncertain whether any of them are the actual cause of grippe, and whether the symptoms are not produced by a micro-organism still undiscovered.

Grippe is very contagious. In the large majority of instances transmission is direct through the infectious secretion from the respiratory tract. Usually an outbreak among the children of a family arises from one of the adult members sick with the disease. The air may also spread the disease by disseminating the dried infected mucus, but probably only to a limited distance. Indirect transmission by a third healthy person or by clothing, and the like, is of doubtful occurrence. The entrance of the germs into the body is by way of the respiratory tract.

Pathological Anatomy.—Apart from the presence of a catarrhal condition of the respiratory and alimentary mucous membranes the lesions are those only of the complications which may arise.

Symptoms.—The clinical manifestations are most varied. We may, however, recognize a *typical form* of grippe, and a number of *modified forms*, marked by the predominance of certain groups of symptoms. Some one or another of the variations has oftener been witnessed than the type itself. In the epidemic of 1918, however, there was less variation seen than in preceding ones.

TYPICAL FORM. Incubation.—This is short, lasting from 1 to 3 days and is generally unattended by symptoms. Occasionally malaise, irritability, vague pains and loss of appetite are witnessed.

Symptoms of the Attack.—In typical grippe there is exhibited a fair balance between the various groups of symptoms which characterize the different varieties. The onset is usually sudden, with chilliness, high fever and sometimes convulsions. In older children pain in the limbs, trunk and head may be complained of; in younger ones there is clearly discomfort, the nature of which cannot be determined. The temperature does not remain persistently high, but runs an irregular and characteristic course, varying from 100° to 105°F. (37.8° to 40.6°C.) and falling by lysis or crisis in 3 to 4 days (Fig. 145). The respiration and pulse are accelerated in proportion to the temperature. Vomiting sometimes occurs and may be obstinate; diarrhea may develop; loss of appetite is marked. Prostration, out of proportion to the other symptoms, is one of the most characteristic manifestations. A varying degree of inflammation of the nose and throat and less often of the trachea and bronchi is present, yet not of a nature sufficient to account for the general symptoms. Albuminuria is seen in a small proportion of cases; leucocytosis is sometimes present, oftener absent; enlargement of the spleen is not infrequently discoverable. In the 1918 epidemic leucopenia was a very characteristic feature. The *duration* of the acute attack is generally only from 3 to 5 days, but decided debility, anorexia and often neuralgic pain may remain for several weeks. The symptoms in early life are generally not so severe as in adults, but to this there are numerous exceptions.

This ordinary type may be either *mild* or *severe*, and between the two extremes all grades of severity are seen. In the *mild cases* local manifestations may be entirely absent and there may be only a very moderate

degree of prostration with slight fever (Fig. 146). The child may not feel ill enough to go to bed, and in a day or two is entirely convalescent. This is a form quite common in early life. In the *severe cases* the temperature may be high or only slightly elevated, but in any event it runs no regular course and is uncharacteristic. The prostration is very decided, appetite is completely lost and some of the localizing symptoms may be marked. Sometimes the course is protracted for weeks or even months, with debility and in older children more or less pain. In infants especially the attack may be very severe, vomiting troublesome,

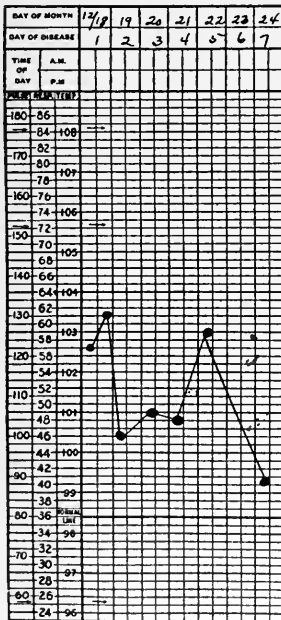


FIG. 145.

FIG. 145.—GRIPPE, TYPICAL FORM.

Anna S., aged 10 months. Cough, running eyes, fever, debility, frequent loose stools, vomited once. Slight preponderance of gastrointestinal symptoms. Epidemic prevailing.

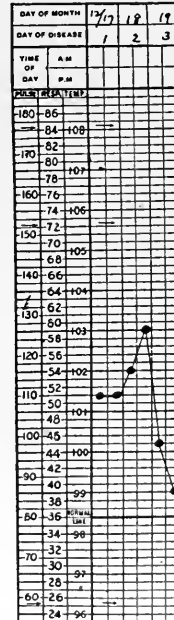


FIG. 146.

FIG. 146.—GRIPPE, TYPICAL FORM, MILD.

Hannah E., aged 4½ years. Vomited occasionally, headache, slight sore throat, loose cough, no marked prostration. Child bright and very little ill. Epidemic prevailing.

loss of appetite absolute, prostration very great, and respiration rapid. The patient may seem to be overwhelmed by the poison of the disease without any evidence of local lesions and collapse may follow. The severe cases last from a few days up to 2 or more weeks, and convalescence is very tedious (Fig. 147).

The different variants from typical grippe depend on the prominence of symptoms of a certain class, and the lesser development of those of other classes. Not infrequently the symptoms of two or more classes may be equally prominent. As a rule the nervous and gastrointestinal forms of grippe are most frequent in infancy and early childhood. The respiratory is that most often seen in later childhood. Yet to this there

are numerous exceptions, and all sorts of combinations of symptoms of the different forms may be seen.

CATARRHAL OR RESPIRATORY FORM.—In this form coryza is decided, the pharynx is red, the tonsils swollen with the follicles sometimes engorged; stomatitis may occur; there is annoying cough depending upon involvement of the larynx, trachea and bronchi; and fever and prostration are present to a degree in no way explained by the local manifestations (Fig. 148). The severity of the attack generally diminishes in 3 or 4 days. In severe cases of this type the course is longer

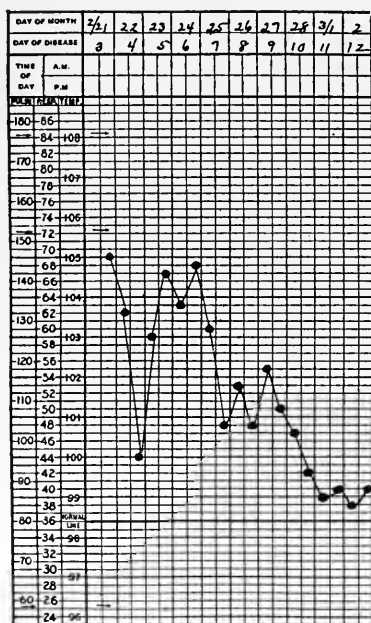


FIG. 147.

FIG. 147.—GRIPPE, SEVERE RESPIRATORY TYPE.

William McC., aged 11 months. Feb. 19, for some days cold in head and moderate bronchitis, no fever; Feb. 21, temperature rose in afternoon, croupy cough; Feb. 22, cyanosis; Feb. 23, many râles, with cyanosis and oppression suggesting asthmatic bronchitis; Feb. 24, seems very ill, cyanosis and râles continuing; Feb. 27, râles nearly gone, general condition improving. Brother ill at the same time with grippe.

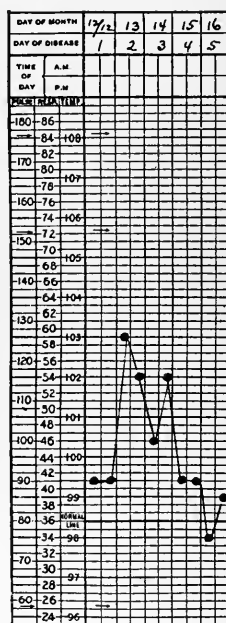


FIG. 148.

FIG. 148.—RESPIRATORY FORM OF GRIPPE.

William R., adult. Headache, general aching, severe coryza, harassing cough, congested frontal sinuses requiring local treatment, marked prostration. Decided debility as a sequel.

and the process readily advances to the production of bronchopneumonia. There is also a special tendency to the development of otitis and of cervical adenitis. The catarrhal type is not often very severe under the age of 10 years, and especially below that of 3 years, but even infants will occasionally exhibit it. (See Fig. 147.) Sometimes the symptoms of this form are unusual and not sufficient to mark the disease except in family outbreaks. I have seen adults affected by characteristic grippe and some of the children of the family showing the symptoms of spasmodic croup (*laryngeal form of grippe*). In the earlier part of the 1918 epidemic catarrhal symptoms, and especially tonsillitis, were decidedly

infrequent. Later, as the severity of the cases grew less-marked, tonsillitis became a very prominent feature.

NERVOUS FORM.—In this variety the nervous manifestations predominate and the respiratory and digestive symptoms are less in evidence (Fig. 149). To this class belong a large number of cases in quite early life. There is marked apathy, prostration and loss of appetite. In some cases hyperpyrexia, delirium, stupor or convulsions may occur, so that the case closely resembles meningitis; in others the symptoms of the typhoid state may develop. Still other cases exhibit marked prostration

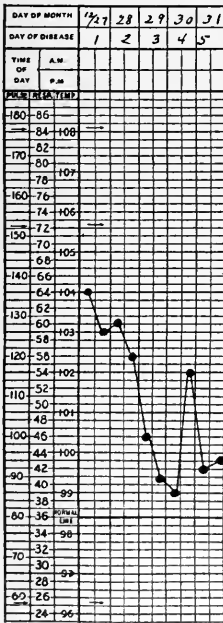


FIG. 149.

FIG. 149.—GRIPPE, NERVOUS FORM.

Helen C., aged 19 months. Pale, prostrated, irregular respiration, very drowsy. No pulmonary symptoms, no diarrhea, vomited only once. Epidemic prevailing.

FIG. 150.—GRIPPE, NERVOUS FORM. PROLONGED FEBRILE TYPE.

Louise F., aged 16 months. Slight cough, apathy, prostration, without any localized symptoms. Fever continued a week or more. Twin sister with same symptoms. Case suggested typhoid fever. Epidemic of gripe prevailing.

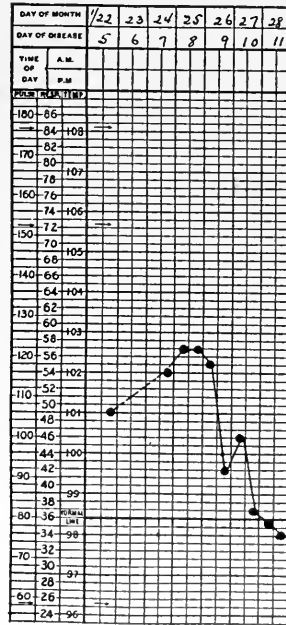


FIG. 150.

and continued fever, which, in the absence of special localizing symptoms at first strongly suggests typhoid fever (*febrile form of gripe*) (Fig. 150). Occasionally there is severe dyspnea of purely toxic origin. Severe cases may sometimes end fatally, although generally in the course of a few days all the alarming symptoms ameliorate, showing that they were toxic and not inflammatory in nature. In other instances actual lesions of the cerebrospinal system develop as complications or sequels. (See Influenzal Meningitis, Vol. II, p. 325.) A very common variety of the nervous type of gripe, especially frequent in younger subjects, is that showing continued fever with moderate prostration, and few distinct local manifestations. Filatow¹ has reported cases of this sort in which irregular fever lasted during 5 months.

¹ Arch. f. Kinderheilk., 1899, XXVII, 433.

GASTROINTESTINAL FORM.—The prominent symptoms of this variety, a common one in infancy, are anorexia, nausea, vomiting, abdominal pain, prostration and diarrhea, the stools often containing mucus and blood. The symptoms are those of a severe acute gastroenteritis. The duration of the attack may be only 3 or 4 days, but is not infrequently protracted. In other cases the condition is one resembling intestinal toxemia, and there are present the ordinary symptoms of this, with loss of weight, but without diarrhea or vomiting. This is a common variety in infancy. The intestinal form was much less frequently observed in the 1918 epidemic than in earlier ones.

Complications and Sequels.—One of the most dangerous and common complications is *pneumonia*. It was especially frequent in the epidemic of 1918, there being an incidence of about 10 per cent. The form is oftenest a bronchopneumonia, the areas being small and often giving no positive physical signs. Less often it is croupous in nature. The pneumonia of grippe develops either during the attack or as a sequel. The course is irregular, the symptoms being often masked and much more severe than accounted for by the physical signs, and the disease frequently of the wandering type and much prolonged. In many other cases it is abortive and lasts only 2 or 3 days. One of the characteristics of the pneumonia in the last epidemic was the tendency often seen for the respiration to exhibit little acceleration; and especially the persistence of leucopenia in spite of the presence of the pneumonic inflammation. (Fig. 151). Pleurisy not infrequently accompanies the pneumonia and tends to result in empyema. *Bronchitis*, constantly present in the respiratory type of influenza, may be severe enough to constitute an important complication, especially in young children. It then involves the smaller tubules and produces dyspnea and cyanosis. Spasmodic croup is a complication sometimes seen.

Otitis, purulent or catarrhal, or often hemorrhagic is a frequent complication especially of the respiratory form of grippe. Either resolution or perforation may occur. Inflammation of the mastoid cells may follow. Affections of the eye are rarely observed except the moderate conjunctivitis which may properly be called a symptom. *Cervical adenitis* is a very striking and frequent complication. It is often out of proportion to the severity of the attack and independent of the presence of affections of the mouth. Resolution is the rule. Parotitis is sometimes observed. Neuritis of various forms may develop as a sequel, although infrequently in children. *Meningitis* depending upon the influenza bacillus or other germs, may accompany the attack, or occur without other symptoms. Adams¹ reported a case, and collected 20 others from literature, in which the bacillus of influenza was found in the cerebrospinal fluid or in the meninges. I have seen several such instances, in none of which, however, could the condition properly be called a complication of grippe. (See *Influenzal Meningitis*, Vol. II, p. 325.) Mental disturbances, spastic paralysis, chorea, encephalitis and other nervous affections exceptionally follow grippe. Nephritis, cystitis and pyelitis are rare. Miller² collected 40 recorded cases of acute hemorrhagic *nephritis* occurring as a complication. Only a few of these were in children. Peritonitis has been reported as a complication or sequel. Ileocolitis may develop in the gastrointestinal form of the disease and icterus is sometimes witnessed. Anemia, at times severe, is a frequent

¹ Arch. of Pediat., 1907, XXIV, 721.

² Arch. of Pediat., 1902, XIX, 1.

sequel. Cardiac complications are few in children, except the tendency to dangerous collapse occasionally observed, dependent at times on acute cardiac dilatation. *Cutaneous complications* are interesting and not rare, especially herpes, urticaria and erythema, which may be either morbilliform or oftener scarlatiniform.

Other infectious diseases may be associated with grippe, among those reported being erysipelas, mumps, scarlet fever, measles, pertussis and diphtheria. The development of tuberculosis as a sequel is not infrequent.

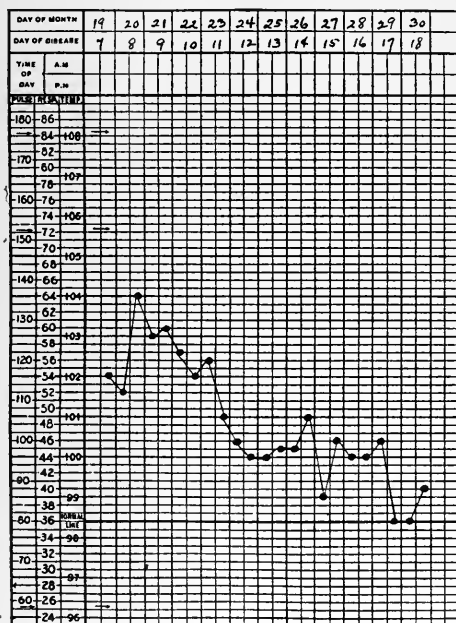


FIG. 151.

FIG. 151.—GRIPPE FOLLOWED BY PNEUMONIA WITH LEUCOPENIA.

Theodore N., aged 3 years. Admitted Oct. 19th, 1918, to the Children's Ward of the Hospital of the University of Pennsylvania. Father and mother and one of the other children of the family ill with grippe. Child developed fever and cough about a week before admission. Examination showed fully developed signs of consolidation, lower part of both lungs; child very toxic and debilitated. Blood on Nov. 4th, showed 7,600 leucocytes. Gradual recovery.

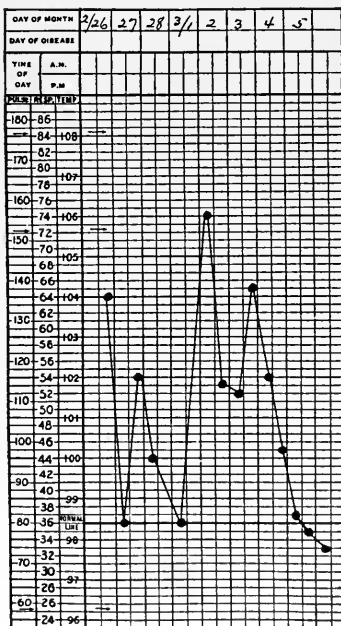


FIG. 152.

FIG. 152.—GRIPPE, RESPIRATORY FORM WITH PROMPT RELAPSE.

Josephine V., aged 6 years. Feb. 26, slight sore throat, high fever; Feb. 28, better, considerable cough; Mar. 1, apparently convalescent; Mar. 2, throat painful, respiration accelerated, cough, high fever, severe coryza; Mar. 4, numerous râles, in other respects better. Mother and brother ill with grippe.

Relapse and Recurrence.—*Relapse*, in the sense of relighting of symptoms before the disease is thoroughly over, is exceedingly common. It develops a few days after symptoms have subsided, possibly after the patient has been taken into the open air, and may equal or exceed in severity the primary attack (Fig. 152).

Recurrence is common. Protection given by one attack of grippe lasts only a few weeks or months, sometimes longer. The large number of individuals developing a second attack a year or less after the first one

shows the uncertainty of immunity. In fact, those who have once suffered appear often especially predisposed later.

Prognosis.—The mortality of uncomplicated grippe has been low, statistics varying from 0.5 per cent. to somewhat over 1 per cent. Nevertheless, when we consider the enormous frequency of the disease, the actual number of deaths from grippe or its complications has reached a surprising figure, and this is particularly true of infancy. Thus, in London alone from January, 1890 to March, 1891, 4740 persons died from it (Leichtenstern).¹ As a rule there is little danger in uncomplicated cases in previously healthy subjects. The chief danger of the disease lies in the complications, especially gastrointestinal disorders and pneumonia, the latter being the cause of the majority of deaths. In the 1918 epidemic the mortality was very high. In 46 of the largest cities of the United States² with a combined population of 23,000,000, the number of deaths from grippe from September 9th to November 8th equalled 82,306, chiefly from pneumonia. The normal number of deaths from these causes for the same period would have been about 4000. In Philadelphia³ during 7 weeks of the epidemic 12,687 deaths were reported from influenza and pneumonia; the number of deaths from the latter disease in the previous year having been only 293. These statistics apply to individuals of all ages. The mortality in childhood as a whole was decidedly less than later in life, although in the 1st year there is a greater tendency to complications, and consequently a greater danger from the diseases, than in the years immediately following.

Diagnosis.—This is easy in typical cases during an epidemic. Under other circumstances it may be difficult or impossible. Prostration, which is present in practically every case except the very mildest, and which is out of proportion to any discoverable cause, is the most important diagnostic symptom. Fever, too, not satisfactorily accounted for in any other way, is another characteristic, as is the pain in many cases. The fact that the mucous membrane of more than one region is affected is also suggestive.

Mild isolated cases of the respiratory type cannot be distinguished from ordinary catarrh of the respiratory tract; the severer ones exhibit too great prostration and fever, and in general are more ill than a simple catarrh explains. The presence of cervical adenitis too, is often suggestive. Cases of the nervous type may present cerebrospinal symptoms suggesting meningitis. The diagnosis is impossible at the beginning, but rapid improvement in the course of a few days, with the development of the more ordinary evidences of grippe make the matter clear. In this connection must be emphasized the necessity of caution in making the diagnosis even of a complicating meningitis, unless this is done by means of lumbar puncture. The protracted febrile cases of influenza without special localized manifestations may be difficult to distinguish from typhoid fever. The suddenness of the onset, the frequent presence of catarrhal symptoms of the nose and eyes, and the absence of the typhoid serum-reaction and of roseola aid in distinguishing it. Enlargement of the spleen may be present in either disease, but is more constant in typhoid fever.

Scarlet fever, measles, and especially pneumonia all bear resemblance to grippe at the beginning. I have seen the erythema which sometimes complicates grippe add to the difficulty in diagnosing between grippe

¹ Nothnagel, *Encyclopedia of Pract. Med.*, Amer. Edit., Influenza, 569.

² Bureau of the Census. Ref., *Med. Rec.*, 1918, CXIV, 906.

³ Monthly Bull., *loc. cit.* 20.

and scarlet fever. The course of the case serves later to distinguish them. Malaria exhibits usually a more regular temperature curve, the spleen is decidedly enlarged, and the malarial organism can be found in the blood.

Gastroenteritis at first resembles exactly the gastroenteric type of grippé. The sudden clearing up of the symptoms will distinguish the latter in the course of a few days. The existence in several members of a family of fever with decided enlargement of the cervical lymphatic glands, an occurrence which has frequently been observed in influenza, sometimes causes the diagnosis of glandular fever to be made. Undoubtedly such mistakes are common. The prevalence of grippé and the presence of undoubted symptoms of it in combination with the glandular enlargement, will generally make the diagnosis evident.

Treatment. Prophylaxis.—The great contagiousness of the disease renders necessary the protection of those not affected, particularly infants and delicate children. This is, however, difficult of accomplishment during epidemics. Certainly the ill child should be isolated as far as possible. It is still better to remove the other children from the house. Quarantine should continue while evidence of respiratory catarrh persists, since living microörganism are probably still present in the secretion. The value of prophylactic vaccines is still undetermined in spite of the careful study which has been given to the subject, and the wide trial of vaccines which was made in the 1918 epidemic.

Treatment of the Attack.—The brief course of the uncomplicated disease and the low mortality usually renders little treatment necessary. There is no specific known and therapy must be symptomatic. What has been said of vaccines for prophylaxis applies equally well here. Rest in bed is imperative. The diet should be light, nourishing and abundant. Fever may be treated by ordinary febrifuges and a laxative, or if high, by sponging or a warm bath. Pain may be relieved by small repeated doses of the coal-tar derivatives, preferably phenacetin and antipyrine, given cautiously. (See pp. 229, 231 for dosage.) Vomiting may be checked by free opening of the bowels and by the administration of bismuth, lime water, bromides or, possibly, opiates. Antiseptic alkaline sprays to the nasal mucous membrane are of advantage. Alcoholic stimulants or strychnine are needed if there is much prostration. Harassing cough may require sedative treatment. Cerebral symptoms may need bromides, coal-tar preparations or opiates. Warm baths combined with cold to the head may prove useful under these conditions.

Complications must be guarded against as far as possible. Exposure to chilling by too early leaving the bed or the room may precipitate a relapse or predispose to pneumonia. Keeping the ears carefully covered during the attack may tend to prevent an otitis. Complications developing should receive the treatment appropriate to them.

In the case where the convalescence is slow and the remaining debility persistent, tonic treatment, careful and abundant diet, and often change of climate may be indicated.

CHAPTER XIV

PERTUSSIS

(Whooping-cough)

History.—The first published recognition of pertussis was by Baillou¹ in Paris in 1578. Willis² observed it in England in 1658 and described it clearly. After the middle of the 18th century the disease spread widely and is now one of the commonest of the acute infectious disorders over the whole civilized world.

Etiology. Predisposing Causes.—Climate, race and geographical position exert no influence, and that of sex is immaterial, females appearing slightly more predisposed. The statistics regarding season are at variance, but on the whole pertussis would appear more frequent in the colder months, possibly through the more intimate association of children in schools at this time. The statistics of Luttinger,³ however, upon 6868 cases showed a greater prevalence during the spring and summer. Poor hygienic conditions favor the dissemination of the disease, but predispose in no other way. The previous state of health is an etiological factor to some extent, weakly and sickly children apparently contracting pertussis more readily than others. The existence of other diseases does not prevent its development, and it is certain that outbreaks of pertussis are especially liable to accompany or to prevail after epidemics of measles, although uncertain whether there is an actual etiological relationship.

Age exercises a powerful predisposing influence. The majority of cases occur under 6 and comparatively few after 10 years. Szabo⁴ reports 1028 cases (20.75 per cent.) in the 1st year out of a total of 4951. Vladimirov⁵ gives the incidence of the disease in 4623 cases as follows:

TABLE 68.—INCIDENCE OF PERTUSSIS AS REGARDS AGE

Under 6 months.....	5.5 per cent.
6-12 months.....	11.1 per cent.
1- 2 years.....	18.1 per cent.
2- 3 years.....	14.2 per cent.
3- 4 years.....	11.6 per cent.
4- 5 years.....	9.1 per cent.
5- 6 years.....	7.9 per cent.
6- 7 years.....	6.1 per cent.
7- 8 years.....	4.7 per cent.
8- 9 years.....	4.1 per cent.
9-10 years.....	3.3 per cent.
10-11 years.....	1.7 per cent.
11-12 years.....	1.3 per cent.
12 years and over.....	1.2 per cent.

Baginsky⁶ found in 2651 cases in children 830 (31.3 per cent.) from birth to 1 year, 1308 (49.4 per cent.) from 1 to 4 years, 502 (18.2 per cent.) from 4 to 10 years, and 11 (0.4 per cent.) from 10 to 14 years. Finally

¹ Geneva Edition, 1742.

² Path. Cerebri, etc., Cap. XII, 1667.

³ Amer. Jour. Dis. Child., 1916, XII, 290.

⁴ Pest. med.-chir. Presse, 1881, No. 33.

⁵ Bolnitch, Gaz. Botk., 1893, No. 12. Ref. O'Dwyer and Norton, 20th Century Practice of Med., XXIV, 217.

⁶ Kinderkrankheiten, 1905, 265.

in an analysis by Luttinger¹ of 10,000 cases the age-incidence was: Under 1 year 19.4 per cent.; 1 to 2 years 20.1 per cent.; 2 to 5 years 40.1 per cent.; 5 to 15 years 17.9 per cent.; 15 years and over 2.3 per cent.

It is evident that the greater immunity of the 1st year seen in some other infectious diseases does not obtain to any extent in pertussis. Even cases of congenital pertussis have been reported (Rilliet and Barthez;² Gatti;³ Cockayne⁴).

The individual susceptibility is very great and the majority of children exposed contract the disease. That it is comparatively uncommon in adults depends largely on the fact that so many have already suffered earlier in life. Some children, however, possess a natural immunity. Epidemic influence is marked, many more cases occurring in some years than in others. The disease is practically endemic in larger cities.

Exciting Cause.—Pertussis has been variously described as a pure neurosis of the medulla or of the nerves which control cough, a simple bronchial catarrh, and a pneumogastric irritation from the pressure of enlarged bronchial lymphatic glands. Its evident infectiousness, however, indicates that it is certainly dependent upon the action of some microbe contained in the respiratory secretions. Very minute bacilli found in the respiratory mucus were reported by Afanasieff,⁵ Czaplewski and Hensel,⁶ Arnheim,⁷ Koplik,⁸ Jochmann and Krause⁹ and others. In 1906 Bordet and Gengou¹⁰ described a very small, ovoid, Gram-negative bacillus present in large numbers in the sputum in the early part of the attack. It resembles, but is quite distinct from, the influenza-bacillus. The discovery has been confirmed by many later investigators, and the etiological relationship of the germ to pertussis is rendered still more positive by agglutination and complement-fixation tests; and by the claims (Klimenko;¹¹ Inaba¹²) that with it the disease could be transmitted to apes and to puppies (Mallory, Horner and Henderson).¹³ While it is very probable that pertussis is due to this germ, further studies are needed, since the agglutination and complement-fixation tests do not always respond positively; there are probably several different strains of the bacillus; the germ may be found in other conditions certainly not pertussis (Fränkel);¹⁴ and cases of pertussis are repeatedly encountered in which no bacterial cause can be discovered, or other bacteria are present, especially those belonging to the group of the influenza-bacillus. The *duration of life* of the germ outside of the body is probably brief. Rooms occupied by the patient soon lose their infectiousness.

Nature of the Disease.—The mode of action of the infectious agent is uncertain. The studies of Meyer Huni¹⁵ and of von Herff¹⁶ indicate

¹ *Loc. cit.*

² Sanné, *Mal. des enf.*, 1891, III, 747.

³ *La Pediatria*, 1914, XXII, 687.

⁴ *Brit. Journ. Child. Dis.*, 1913, X, 534.

⁵ *Petersburg med. Wochenschr.*, 1887, IV, 323.

⁶ *Deutsch. med. Wochenschr.*, 1897, XXI, 586.

⁷ *Berl. klin. Woch.*, 1900, XXXVII, 702.

⁸ *Brit. Med. Journ.*, 1897, II, 1950.

⁹ *Zeitschr. f. Hyg.*, 1901, XXXVI, 193.

¹⁰ *Annal. de l'Institut. Pasteur*, 1906, XX, 731.

¹¹ *Deut. med. Woch.*, 1908, XXXIV, 203.

¹² *Zeit. f. Kinderh. Orig.*, 1912, IV, 252.

¹³ *Journ. Med. Res.*, 1913, XXVII, 391.

¹⁴ *Münch. med. Woch.*, 1908, LV, 1683.

¹⁵ *Zeitschr. f. klin. Med.*, 1880, I, 461.

¹⁶ *Deut. Arch. f. klin. Med.*, 1886, XXXIX, 392.

that the seat of irritation is oftenest the nose, larynx and trachea, but especially the inter-arytenoid fossa on the posterior wall of the larynx—the so-called “cough region.” It is probable, however, that the situation of the local irritation upon the respiratory mucous membrane may vary to some extent with the case. As a result of this local irritation a paroxysm of cough is produced by the accumulated mucus upon the sensitive region. In this way is set going a series of reflex clonic spasms of the respiratory muscles, the whoop being due to an inspiratory spasm of the glottis. The process is repeated until the offending mucus is expelled. From this point of view the disease is chiefly a local irritation produced by an infectious catarrhal process. It seems, however, an unavoidable conclusion that, in addition to the local irritation which precipitates the paroxysms, there exists a general constitutional disorder which determines the peculiar character of these, and that this character may depend upon a disturbance of the superior laryngeal nerves and the respiratory centres in the medulla, brought about by a toxin produced by the germs and circulating in the blood. This view is supported by analogy to other infectious diseases; the production of attacks of cough by excitement and other disturbances acting reflexly; the greater frequency of the cough at night indicating lesser resisting power of the respiratory centres in the medulla; the occasional occurrence of congenital pertussis, which must necessarily be a blood-disorder; the much increased excitability of the general nervous system which is always present; the occurrence of an agglutinative reaction; etc.

Period of Greatest Infectiousness.—This is not certainly determined. There is no question that the disease is transmitted early in the catarrhal stage, and perhaps most decidedly at this time. There is also no doubt, on the other hand, that it can be transmitted in the paroxysmal stage and even during the decline.

Mode of Transmission.—This is direct by the secretion of the respiratory tract and to a certain extent by the breath, the germs being contained in minute droplets of mucus expelled by coughing. Conveyance to any distance by the air does not appear to occur. Only exceptionally is the infection carried by a third person; and it is very probable that in most of such supposed cases the disease has in reality been communicated by someone suffering from an abortive and unrecognized attack. The germs are received through the respiratory tract, the congenital cases of pertussis being the exception.

Pathological Anatomy.—There are no characteristic lesions. During life there is found an intense redness and swelling of the respiratory mucous membrane, especially of the larynx and trachea, and to a less extent of the nose and pharynx, with the secretion of viscid mucus. At autopsy involvement of the bronchial mucous membrane may also be discovered, and very constantly some degree of pulmonary emphysema. Mallory and Horner¹ found the Bordet-Gengou bacillus between the cilia of many of the cells of the mucous membrane of the trachea and bronchi. Acute bronchiectasis, enlargement of the bronchial and tracheal glands, and congestion of the brain, lungs and other internal organs are often found. Some of the lesions of complicating conditions are generally present in fatal cases, among these being bronchopneumonia, atelectasis, cerebral hemorrhage, hypertrophy and dilatation of the heart, especially the right ventricle, and hemorrhages in various organs, especially the brain.

¹ Journ. of Med. Res., 1912, XXVII, 115.

Symptoms.—The attack is usually divided into four periods: (1) the incubation; (2) the invasion or the catarrhal stage; (3) the paroxysmal, spasmodic, or convulsive stage; (4) the stage of decline. These are not very sharply differentiated and vary greatly in length in different cases.

Incubation.—Owing to the insidiousness of the invasion the exact *duration* of incubation is difficult to determine, but it probably varies from 2 to 14 days with an average of 3 to 4 days. No symptoms are present.

The Catarrhal Stage.—The attack begins with the symptoms of a tracheobronchitis, often with some degree of coryza, sneezing, hoarseness and pharyngeal irritation. Slight elevation of temperature with malaise and irritability may be present at the beginning and last a few days. The cough at first is in no way suspicious, but later becomes hard, dry and annoying. In typical cases the chest reveals few if any râles, but sometimes an attendant bronchitis obscures the symptoms and may be the cause of continued fever. The cough gradually becomes more severe, frequent, and paroxysmal in character, and attacks are especially prone to occur in the night. Finally, distinct whooping develops and the paroxysmal stage may be said to have fairly begun. The condition of the blood will be referred to later.

The *duration* of the catarrhal stage is extremely variable. Some children begin whooping after 2 or 3 days, others only after 3 or 4 weeks, or not at all. In general it averages about 2 weeks, but the younger the child the shorter this period.

The Paroxysmal Stage.—The beginning of this period is usually dated from the commencement of whooping. A typical paroxysm is very characteristic. Often it comes on without warning, but often, too, the child experiences a slight tickling in the throat or beneath the sternum, an inclination to cough, or a sensation of smothering or of intense anxiety. If previously lying down it sits upright with an anxious expression and perhaps grasps the side of the crib. If moving about it drops its toys and runs to its mother or nurse, or takes hold of some of the furniture of the room. There is a brief moment of holding the breath, a deep inspiration follows, and the attack begins. This consists of a series of short explosive coughs, so rapidly repeated that there is no time for respiration between them. These continue for a few moments and number anywhere from 4 or 5 up to 15 or 20. Meanwhile the face becomes swollen, red, cyanotic, and sometimes quite dark and the eyes prominent and congested; the tongue protrudes with each expiratory effort; tears flow from the eyes and saliva from the mouth; the veins of the neck are engorged; perspiration breaks out on the face, and the pulse increases in frequency. Finally the cough ceases and the respiration often apparently also, but in a moment a long-drawn, crowing inspiration is heard which is called the "whoop," and depends upon a spasm of the glottis. Immediately after the whoop a second attack generally occurs and after this perhaps a third or fourth, or more. The whole paroxysm lasts from a few seconds up to several minutes. In the latter case there may be a momentary period of rest between some of the attacks. Toward the end of the paroxysm very tenacious, ropy mucus is often driven from the mouth by the force of the cough and this seems to bring relief, and retching or vomiting is liable to follow.

The paroxysms vary from 6 or 8 up to 60 or more in 24 hours, not all of them in severe cases being equally marked. They are generally most

troublesome at night. They occur without discoverable reason, or are brought on by such slight causes as excitement, crying, swallowing, exercise, sudden change of air, inhalation of the air of close rooms, the use of a tongue depressor, hearing another child in a paroxysm, and the like. The presence of mucus on some part of the irritated mucous membrane is the commonest immediate cause. Auscultation of the chest during the expiratory efforts reveals no respiratory sound and only the impulse of the cough against the ear. During the whoop only a feeble inspiration is heard or none at all. In severe cases the urine and feces may be involuntarily expelled and hemorrhage take place from the nose or mouth or beneath the conjunctiva.

More or less fatigue, usually of brief duration, may follow the paroxysm. In severe cases the child may be covered with perspiration after an attack, confused, and quite exhausted. Between the paroxysms a characteristic appearance of the face is often seen, consisting of some degree of swelling and cyanosis, congestion of the eyes, and blueness of the tongue.

The urine in pertussis may contain albumin in severe cases. A slight blowing apical murmur may be heard, and on percussion the cardiac dullness may be increased (Koplik).¹

The *blood* as studied by Fröhlich² and others, and more recently by Crombie,³ Kolmer,⁴ McGay,⁵ and others, shows a remarkably high leucocytosis, averaging 20,000, and often reaching higher figures, which begins early in the catarrhal stage and reaches its maximum at the height of the paroxysmal stage. The increase is seen in the lymphocytes, the neutrophiles being relatively diminished. The condition of the blood returns to normal in 2 or 3 months, the neutrophiles gradually increasing and the lymphocytes diminishing. The eosinophiles are diminished (Benetz).⁶ In the severest cases the leucocytosis is greater, while the lymphocytes are fewer and the neutrophiles more numerous than in those of average severity (McGay; Crombie).

The severity of the paroxysmal stage increases for about 2 weeks, and then continues unabated. Meanwhile the general nutrition and strength are but little affected in average cases in previously healthy children; the appetite and digestion are good, and there is no fever unless complications are present. In severe cases, however, the child may suffer greatly from lack of sleep and loss of food by vomiting, and emaciation and debility may become extreme. As the disease advances a few moist râles become audible between the paroxysm.

The *duration* of the paroxysmal stage is very variable. It averages 3 to 6 weeks, but may be much longer, while in the mildest cases this stage may continue not more than a week.

This description of the typical spasmodic stage does not apply to all cases. Sometimes the paroxysms are so mild or so infrequent that the child is little disturbed or perhaps needs relief only during the night, if at all. In some cases no whooping occurs at any time and the other symptoms are but little marked. It is likely that these very mild cases are often unrecognized and are the means of the dissemination of the disease. In others, especially in the 1st year of life, the whooping may be

¹ Trans. Amer. Pediat. Soc., 1893, V, 90.

² Jahrb. f. Kinderh., 1897, XLIV, 53.

³ Edinb. Med. Journ., 1908, I, 222.

⁴ Amer. Jour. Dis. Child., 1911, I, 431.

⁵ Cleveland Med. Journ., 1911, X, 571.

⁶ Nederl. Tijdschr. v. Geneesk., 1910, LX, 153.

replaced by dangerous apnea, with unconsciousness. In some instances the attack may be accompanied by violent repeated sneezing, and exceptionally this may entirely replace the cough (Szego).¹ The paroxysmal character of the second stage may also be influenced by complications. It may, for instance, disappear completely for a time if pneumonia develops.

Stage of Decline.—This stage is naturally not sharply separated from the preceding one. Its beginning is marked by a lessening in the number and frequency of the attacks. This steadily continues, the cough growing looser and losing more and more its peculiar character, while the whooping follows only some of the paroxysms. Moist râles are heard in greater numbers in the chest and the sputum is more purulent. Finally whooping ceases entirely and the disease is over, although more or less cough may continue for an indefinite time. The *duration* of this stage is even more variable than that of the others. In general it may be said to average from 2 to 3 weeks. It may, however, be indefinitely prolonged by the development of a slight bronchitis, which may cause a return of the severity and an increase in the number of paroxysms. Consequently during the winter season the paroxysmal stage is liable to be prolonged.

It not infrequently happens that the whooping returns after it has ceased entirely for a short time. This cannot properly be called a continuation of the disease, but is rather now a pure neurosis without any infectious element. The actual termination of the attack of pertussis in average cases may generally be placed at the time when the whooping and the paroxysmal character of the cough have entirely disappeared for a number of days. The total duration of the disease thus equals from 6 weeks to several months.

Complications and Sequels.—*Respiratory* complications and sequels are the most frequent. A moderate bronchitis is a symptom of the stage of decline. If it is unusually severe or develops early in the attack, it is to be regarded as a complication. It is serious if involving the small tubes, or when occurring in the 1st year of life. The most common and one of the most dangerous complications is bronchopneumonia. The younger the child the greater the predisposition to it. It is also especially frequent in feeble or rachitic subjects. It makes its appearance oftenest during the height of the convulsive stage or later. During its presence the whooping character of the cough is liable to lessen or disappear. The proportion of cases of pertussis attacked by bronchopneumonia varies. Of 1731 cases of pertussis in the Metropolitan Asylums Board's Hospital in 1912, 10.43 per cent. developed bronchopneumonia (Rolleston).² It has reached even as high as 33.3 per cent. (Sée).³ Atelectasis is of common occurrence in severe cases in infants. A moderate degree of pulmonary emphysema is probably always present, and occasionally remains as a sequel. Pneumothorax has been reported, and croupous pneumonia and pleurisy with serous or purulent effusion are occasionally seen.

Of *digestive disorders* the most troublesome complication is vomiting, which may be so severe and frequent after the paroxysms that great emaciation and loss of strength develop rapidly. Loss of appetite, indigestion, and diarrhea are very often observed in infants, especially in summer, when ileocolitis also is liable to occur. Prolapse of the rec-

¹ Arch. f. Kinderh., 1900, XXIX, 186.

² Brit. Jour. Child. Dis., 1914, XI, 38.

³ Arch. gén. de méd., 1854, II, 279.

tum or hernia may be produced by the violence of the cough, or ulceration of the frenulum linguæ from the repeated impinging of the tongue against the lower incisor teeth. Stomatitis is common in severe cases.

Nervous complications and sequels are frequent, among the most important being general convulsions, which not rarely end fatally. They are oftenest seen in rachitic infants, but occur also in the course of pneumonia or as an indication of intracranial hemorrhage. They are most liable to develop in severe cases, but it frequently happens that fatal convulsions attack without warning an infant who has been apparently little ill. They may occur in any period of the disease. Spasm of the glottis is another convulsive complication sometimes fatal. Aphasia, blindness, deafness and various psychoses are occasional sequels. They are generally of temporary duration, but sometimes permanent. Coma or a soporose condition may occur as a result of intracranial disturbance. Different forms of paralysis, temporary or lasting, are not infrequent sequels. They have been studied with especial care by Valentin¹ and by Hockenjos.² In the majority of instances the paralysis is central in origin and of a hemiplegic type, or occasionally monoplegic or paraplegic, and depends upon cerebral hemorrhage following the violent congestion; or, in cases which recover, sometimes upon temporary passive congestion and edema. Myelitis and multiple neuritis are uncommon. Disseminated sclerosis has been reported as a sequel.

Of *cardiovascular complications* one frequently seen is dilatation of the heart, especially the right ventricle, which occurs in severe cases (Koplik).³ Degenerative changes in the cardiac muscle aid in producing it. Sudden death may result. Hemorrhages are of frequent occurrence, dependent upon the intense passive congestion which the violent coughing occasions. Epistaxis is the most common form and may occasionally be severe enough to require treatment. Discharge of blood from the mouth is oftenest dependent upon epistaxis, the blood having sometimes been swallowed and then vomited. Hematemesis, the result of gastric hemorrhage, is unusual, as is hemorrhage from the lungs or ears or into the skin. Sub-conjunctival hemorrhage is frequent and sometimes so extensive that the entire white of the eye is replaced by a blood-red color. The most dangerous, although not frequent, form of hemorrhage is that within the cranium, oftenest meningeal in nature. The effusion of blood may be small and disappear without permanent injury, or large enough to produce death in convulsions, or to leave lasting paralysis of some sort.

Otitis media is a not infrequent complication. Albuminuria is often seen during the attack and nephritis is an occasional complication or sequel. Glycosuria may develop. Cutaneous emphysema is a rare occurrence.

Other infectious diseases, may follow, precede or accompany pertussis, among them being varicella, diphtheria, typhoid fever, rubella, scarlet fever, grippe, and especially measles. Tuberculosis in some form, especially as bronchial or mesenteric adenitis or as tubercular bronchopneumonia, is a common and dreaded sequel.

Relapse.—As already pointed out, the cough of pertussis has a very great tendency to return after a brief interval through the action of slight bronchitis. In the strict sense this is rather a neurosis than a true re-

¹ Thèse de Paris, 1901.

² Jahrb. f. Kinderheilk., 1900, LI, 426.

³ Loc. cit.

lapse. It sometimes happens, too, that the symptoms of the disease reappear after an interval of weeks or months. Whether this is to be classified as a true relapse is uncertain.

Recurrence.—This is very rare, one attack almost always giving permanent immunity. Errors in diagnosis account for many supposed second attacks. Le Gendre writing in 1891¹ could find but 9 recorded cases of recurrence, including 1 observed by himself. Widowitz² in 558 cases of whooping cough in children found no instance of a second attack. It is probable that this does not apply so strictly to adult life, and that the immunity may occasionally become exhausted at this period. I have seen a few instances in which mothers of children with pertussis developed a modified second attack, and Widowitz records 7 instances of second attacks of the disease in individuals over 30 years of age.

Prognosis.—Contrary to the widespread popular opinion pertussis is a serious disease. Approximately 100,000 children died of it in the United States during 10 years (Johnston)³ and 65,381 died in England and Wales during 8 years (Sticker).⁴ Many additional deaths which have been assigned to complications or sequels might properly be added to these figures. Crum⁵ estimated that 1 per cent. of the total deaths from all causes in 24 countries depended upon pertussis.

Not only is the actual number of deaths from pertussis large, but the case-mortality is very considerable, varying according to different statistics from 3 to 15 per cent. or over. The general mortality from it in Philadelphia during 5 years equalled 6.9 per cent. (Graham).⁶ Age is a powerful factor in effecting this, the danger being the greater the younger the child. In infants pertussis is a dangerous affection, the mortality reaching probably 25 per cent. According to Neurath⁷ the mortality in 6469 cases in Vienna was divided as follows:

TABLE 69.—AGE AND MORTALITY IN PERTUSSIS

Age	No. of Cases	Mortality
1st year.....	1242	25.3 per cent.
2 to 5 years.....	3139	6.8 per cent.
6 to 10 years.....	1926	3.9 per cent.
11 to 15 years.....	135	7.4 per cent.
After 15 years.....	27	0 per cent.

Luttinger⁸ places 97 per cent. of the deaths as under 5 years. After the age of 6 years death is uncommon.

The danger depends much more on the *complications and sequels* than on the disease itself. Weakly and marantic or rachitic infants are especially liable to succumb. The combination of scarlet fever or diphtheria with pertussis, or the immediate precedence of measles, increases the danger decidedly. Obstinate vomiting is a cause of death through the marantic state which results, and tuberculosis is a not infrequent fatal sequel. Often asphyxia following a severe paroxysm is the immediate cause of a fatal issue in infancy. By far the greatest number of deaths, however, depend upon the development of diarrheal diseases,

¹ Rev. mens. de mal. de l'enf., 1891, IX, 496.

² Wien. klin. Wochenschr., 1909, XXII, 1596.

³ Arch. of Pediat., 1885, XII, 241.

⁴ Nothnagel's Encyclop. of Pract. Med. Amer. Ed. Pertussis, 548.

⁵ Amer. Journ. Public Health, 1915, V, 994.

⁶ Journ. Amer. Med. Assoc., 1917, LXVII, 1272.

⁷ Pfaundler and Schlossmann, Handb. d. Kinderkr., 1906, I, 2, 871.

⁸ Loc. cit.

convulsions and, especially, bronchopneumonia, the last mentioned being particularly serious when complicating whooping-cough. Probably over $\frac{1}{2}$ the fatal cases of pertussis are due to pneumonia. Convulsions or pneumonia is especially liable to develop if rickets and pertussis are combined.

Diagnosis.—The diagnostic symptoms in typical cases consist in the evidence of infection; the gradual development of cough which becomes more and more paroxysmal and is finally followed by a whoop; the congestion of the eyes, vomiting, and cyanosis; the tendency for the cough to be worse during the night; and the absence of fever and of physical signs of bronchitis commensurate with the severity of the symptoms. This group of symptoms usually makes the diagnosis simple. In order to hear the cough himself the physician can sometimes produce a paroxysm by examining the throat of the patient with a tongue depressor or by tickling the nasal mucous membrane.

Yet so many exceptions occur that the recognition of pertussis often becomes difficult or impossible. Early in the attack the nature of the disease cannot be recognized with certainty. Except for the history of exposure, it is only through the cough becoming more paroxysmal as time passes, without evidence of increasing bronchitis, that the case becomes suspicious. Vomiting may occur after severe coughing from other causes, and occasional whooping, too, is not uncommon in children, and especially in infants suffering from severe bronchitis. Yet the combination of fever, shortness of breath and numerous râles in the chest constitute sufficient reason to exclude pertussis. The failure of the whoop to develop renders the diagnosis uncertain, unless the other characteristics are well marked and there is distinct evidence of infection. In mild cases not only the whoop, but vomiting and the violent paroxysmal nature of the coughing, is absent or but little marked. Yet even in such cases the prolonged course and its unyielding character to the ordinary treatment of tracheobronchitis renders the case suspicious especially if an epidemic is prevailing; and the diagnosis is practically certain if typical cases develop in other members of the family. In severe cases in infancy the occurrence of cough without whoop but followed by attacks of apnea is strongly suggestive of pertussis. After an attack of measles the diagnosis may at first be especially difficult, since a persistence of the bronchitis with fever may mask the ordinary character of the catarrhal stage of pertussis. A prolonged terminal stage of pertussis may arouse the suspicion of tuberculosis of the lungs occurring as a sequel. Continued observation of the case, and especially the absence of fever, will settle the diagnosis. Tuberculous or simple inflammation of the tracheal or bronchial lymphatic glands may produce a very paroxysmal cough strongly suggesting pertussis. The long continuance of the glandular affection, and the absence of distinct stages, of vomiting, and of well-marked whooping, tend to exclude the latter. An examination of the blood may be an important diagnostic aid, inasmuch as the leucocytosis, especially of the lymphocytes, is higher than in other afebrile affections which could be confounded with whooping-cough. The agglutination test may be of diagnostic value early in the spasmodic stage (Povitzky and Worth);¹ but later the complement-fixation is likely to be of more service. Olmstead and Luttinger² concluded that the latter is positive in 40 per cent. of the cases at the height of the disease or during its decline.

¹ Arch. Int. Med., 1916, XVII, 279.

² Arch. Int. Med., 1915, XVI, 67.

Treatment. Prophylaxis.—In view of the great infectiousness of pertussis and the danger especially in infancy and early childhood, every care should be taken in the separation of those with the disease from others. This should continue at least for 6 weeks in all, or longer if the whooping persists. Although the infectiousness may generally be considered over when once the whooping has stopped for a short time, it is better that the isolation should continue for 2 or 3 weeks after this event. Since the patient is not confined to bed, and cannot properly be kept in one room, the unimmune children of the family should, if possible, be sent away from the house, or, still better, from the locality where pertussis is prevailing. This is especially important in the case of those less than 2 years old and in delicate children of any age. Fumigation and disinfection of the house and its contents are not essential, but are nevertheless a wise precaution. (See also Vaccine Treatment, p. 494.)

Treatment of the Attack.—The number of remedial measures which have been tried is enormous. Some are useless; others good in one case; others in another. Many children require practically no treatment; others tax all the resourcefulness of the physician. A review of some of the methods recommended may be given.

(A) *Hygienic Treatment.*—Unless some complication interferes, confinement to bed is not necessary and the children should be kept in the fresh air as much as possible. This does not mean, however, that they should be sent out of doors regardless of the state of the weather. Damp and windy weather and all chilling of the surface of the body are to be avoided on account of the danger of producing bronchitis or pneumonia. This is especially true in infancy and early childhood, and at this period of life during the winter season the airing is best done in the room with the windows open. Should any bronchitis exist, young patients must be kept in airy rooms frequently and carefully ventilated, and exposure to the air outside must be given cautiously if at all during the cold season. It is an excellent plan to have two rooms, one for the day and one for the night, each being constantly open to the air when unoccupied. The sleeping in a freshly aired room undoubtedly diminishes greatly the number of paroxysms. Clothing should be sufficiently warm and food should be nutritious and easily digestible, and administered in small quantities and frequently when vomiting is troublesome. In such cases it is a good plan to employ a liquid diet, given immediately after the attacks of vomiting. Nutrient enemata are occasionally required in older subjects. Change of air, especially to the seashore, is often wonderfully efficacious, particularly in cases at all prolonged.

(B) *Local Medication.*—This has been largely employed, chiefly on the ground that the disease was a local infection. It may be divided into (a) Insufflation of powders into the nose and larynx; (b) Application of solutions by the spray or brush or by irrigation; (c) Inhalation of volatile substances or of gases.

(a) Quinine has been mixed with boric acid, acacia, or bicarbonate of soda and insufflated in powdered form into the larynx or nose 2 or 3 times a day. Resorcin, salicylic acid, benzoin, tannic acid, and iodoform are prominent among the other numerous drugs which have been used in this way. In my own experience this method of treatment is inferior to internal medication. It is certain, too, that in the hands of physicians not specially trained in making laryngeal insufflation the medicament employed usually does not reach the larynx. When it does, a threatening spasm of the glottis may readily be produced.

(b) A 1 per cent. solution of resorcin applied to the larynx or to the pharynx with the spray or the brush has been much recommended. A solution of cocaine, 2 per cent. or stronger, applied by the brush certainly relieves, but is distinctly dangerous. Prominent among the other numerous drugs have been used in this manner may be mentioned bromide of potash, nitrate of silver, chloride of ammonium, tannic acid, peroxide of hydrogen, and salicylic acid. Irrigation of the nares has often been advocated, among the remedies employed being peroxide of hydrogen, boric acid, sulphate of iron (1 grain : 1 ounce) (0.065 : 30) bichloride of mercury (1:6000) and salicylic acid (1:1000). The treatment is unpleasant and offers no advantages over spraying.

(c) Inhalations of gaseous or volatile substances have certainly often been helpful. One of the most efficacious is carbolic acid or creasote, which may be vaporized from a strong solution in a croup kettle, inhaled from cotton in a respirator placed over the mouth and nose, or volatilized on metal by direct heat from a small lamp. The possibility of absorption and consequent poisoning must be borne in mind. Turpentine, benzine, thymol, oil of cypress, camphor, naphthalene, and eucalyptus are some of the other drugs recommended for inhalation. Anesthetization with chloroform or ether has been advised in exceptionally severe attacks. The value of the fumes of burning sulphur has also been urged. The room is fumigated after the child has left it in the morning, aired after several hours, and slept in at night. Formaldehyde preparations have also been highly recommended for inhalation, and good results have been reported with ozone.

(C) *Systemic Medication.*—This method is, in my experience, more reliable and convenient than that just described. What drugs will be of benefit depends partly on the stage of the disease and partly on the individual reaction of the child. In the initial stage such sedative treatment as is useful in acute tracheobronchitis will often answer, while in the stage of decline expectorants may be indicated, or drugs exhibited to check excessive secretion. The age of the patient is a factor also, general and cardiac stimulation being especially needed in infancy.

Of the very numerous drugs employed only a few have proven useful in the hands of many physicians. First to be mentioned as one of the best is antipyrine. Shortly after its high recommendation by Sonnenberger¹ I began its administration with excellent results.² Children tolerate it in relatively large amount. An initial dose at 3 months may be $\frac{1}{4}$ grain (0.016) which in severe cases may be rapidly increased to $\frac{3}{4}$ grain (0.049) or even 1 grain (0.065) every 3 hours. At 2 years 2 to 3 grains (0.13 to 0.194) every 3 hours may be given. It is practically only when fever is present, due to complications, that antipyrine may exert a depressing effect, and in such conditions it should not be used. The favorable results in some cases seem little short of miraculous. In others it is of no avail whatever. Tussol, a combination of antipyrine with hydrocyanic acid, has been highly praised in doses of $\frac{1}{2}$ to 5 grains (0.032 to 0.324) according to the age. Bromoform, recommended by Stepp³ is often very useful. To a child of from 3 to 6 years it may be given 3 or 4 times a day in doses of from 2 to 10 minims (0.123 to 0.616) on moistened sugar. The initial dose should be increased cautiously, and if the drug causes drowsiness it should be abandoned.

¹ Deutsch. med. Wochenschr., 1887, XIII, 280.

² Therap. Gaz., 1888, Feb., 84

³ Deutsch. med. Wochenschr., 1889, XVII, 639.

Belladonna is an old time favorite often very effective. A child of 2 years may begin with 2 minims (0.123) of the tincture or $\frac{1}{1500}$ grain (0.0004) of atropine 3 times a day. The amount may usually be rapidly but carefully increased, since full doses are generally required.

Quinine has been proven of value in many cases, but often deranges the digestion. The amount required is generally large, 1 grain (0.065) or more every 2 to 4 hours at 2 years of age. The modern comparatively tasteless derivatives of quinine, such as euchinin and aristochin, have been employed successfully to replace it. The bromides are often serviceable in combination with antipyrine and belladonna. Chloral, too, is useful, especially in producing sleep at night. It must, however, be given cautiously to young children on account of its depressing influence. Opium, or its derivatives, is often of great value, particularly if given in a single full dose at night, or sometimes in small doses during the day. Phenacetin, acetanilid and other drugs of this class are sometimes used in place of antipyrine, and are occasionally effective when this fails.

Among the numerous other drugs recommended, for the value of which there is distinctly trustworthy testimony, may be mentioned turpentine, castanea, drosera or droserin, cannabis Indica, asafetida, fluoroform, quebracho, camphor, hyoscyne, grindelia, eulatin, adrenalin, veronal, and thyme or its derivative pertussin. Antitussin, an ointment containing difluorphenyl, although used locally by inunction on the thorax, is a systemic remedy in its action. It seems to be undoubtedly of benefit in some instances.

(D) *Mechanical and Miscellaneous Treatment.*—Here may be placed a number of methods of treatment not already discussed. Prominent among these is the employment of vaccination with anti-smallpox vaccine, the usefulness of which Italian physicians have often maintained. As the occurrence of other diseases sometimes modifies the symptoms of pertussis, vaccination may possibly act in this way. I have personally never been convinced of its value. A modification proposed by Violi¹ consists in the subcutaneous injection of serum from vaccinated heifers. Fitting the child with an elastic abdominal belt has been highly recommended by Kilmer² for the control especially of the vomiting attending the paroxysms, and its good effect has been maintained by others. The employment of the constant galvanic current to the neck and spine and the use of the pneumatic cabinet have each had their advocates. Intubation was tried by O'Dwyer³ in very severe cases with remarkable relief. Nägeli⁴ maintained that the pulling of the lower jaw downward and forward would abbreviate or mitigate the paroxysm, and this statement has been corroborated by Sobel.⁵

A few general remarks may be made on the forms of treatment described. Beginning any of them too early in the attack is to be avoided, since at the most they are symptomatic, and intended only to relieve the severe attacks of coughing. We cannot expect, as a rule, to curtail by them the duration of the disease. Mild cases, in which the paroxysms are few and the general health excellent, may require no therapy at all other than hygienic, or at most, a sedative, such as opium or antipyrine, given at night to lessen the cough and insure sleep. The

¹ Gaz. hebdom., 1897, XLIV, 904.

² New York Med. Journ., 1903, LXXV11, 1101.

³ 20th Cent. Prac. of Med., XXIV, 213.

⁴ Corresp'bl. f. Schweiz. Aerzte, 1889, XIX, 417.

⁵ Arch. of Pediat., 1903, XX, 418.

child should be spared medication when there is no real demand for it. Certainly remedies should not be given which disturb the digestion, since the general nutrition is so liable to suffer in many instances. Further, to determine the value of any treatment for the relief of the paroxysms it is necessary that it be given at the *height of the disease*. Remedies employed toward the end of the paroxysmal stage may appear to do good, only because the severity is naturally lessening by this time. Again, before assuming that a remedy is useless it should be given in sufficiently large dose, of course with careful watching. If it proves of no avail in a few days, we should not abandon our efforts but try something else, since no one method can be equally good in all cases. Complications require measures appropriate to them.

(E) *Vaccine Treatment*.—Various efforts have been made to control the disease by sera and vaccines. Sylvestri¹ employed the blood-serum from convalescent cases of pertussis, and Leuriaux² and Klimenko,³ that obtained from inoculated horses. The greatest interest centers around the employment of vaccines of the Bordet-Gengou bacillus. While the reports of many investigators are most encouraging, and this is especially true of its use as a prophylactic measure (Hess),⁴ the experience of others is opposed, and it is much too early to draw any positive conclusions. Luttinger⁵ and Shaw,⁶ for instance, claimed excellent results, while Von Sholly, Blum and Smith⁷ could find no satisfactory evidence that the vaccine treatment possesses any value. As already pointed out, the Bordet-Gengou bacillus is not found in all cases of pertussis, and in these the disease would appear to depend in some instances upon other microorganisms, and vaccines of the former could not possibly be of benefit. The treatment, however, appears to be harmless and may well be tried, especially in infancy, since at this period the disease is most to be feared. The dosage recommended varies from 25,000,000 to 500,000,000 and over this, repeated every day or every 2 or 3 days.⁸

CHAPTER XV

MUMPS

(Epidemic Parotitis)

The title Epidemic Parotitis is only a partially satisfactory one, for, although the parotid glands are the usual seat of the disease, the other salivary glands may be secondarily, or occasionally primarily or even solely involved, or the disorder may be represented merely by an orchitis. The affection was well described by Hippocrates. Although confounded later with other disorders it was again clearly differentiated toward the end of the 18th century.

¹ Gaz. degl. Osp., 1901, No. 14. Ref., Münch. med. Woch., 1901, XLVIII, 2020.

² La sem. méd., 1902, XXII, 233.

³ Arch. des sciences biol. de St. Petersb., 1912, XVII, 103.

⁴ Journ. Amer. Med. Assoc., 1914, LXIII, 1007.

⁵ Journ. Amer. Med. Assoc., 1917, LXVIII, 1461.

⁶ Pediatrics, 1917, XXIX, 205.

⁷ Jour. Amer. Med. Assoc., 1917, LXVIII, 1451.

⁸ See Review by Beifeld, Amer. Jour. Dis. Child., 1916, XII, 177.

Etiology. Predisposing Causes.—Age has a powerful predisposing influence, attacks being most frequent at from 5 to 15 years, and not uncommon in young adults. It is infrequent under 2 years. Ringberg's¹ statistics based on 58,331 cases in Denmark gave:

TABLE 70.—INCIDENCE OF MUMPS

Under 1 year of age.....	205 cases, 0.35 per cent.
1 to 5 years of age.....	4,512 cases, 7.74 per cent.
5 to 15 years of age.....	12,103 cases, 20.85 per cent.

Even cases in the new born have been reported (Gautier,² 13 days; Demme,³ 2 weeks; White,⁴ 7 days), and it is even possible for the infection to be acquired during fetal life (Homan).⁵ The youngest case coming under my observation was in an infant of 10½ months.

Sex has been claimed to be a factor, males being oftener attacked than females, but its influence is very questionable. Race and climate exert no influence, and the disease is widely spread over the earth. It is most prevalent in the colder season. *Epidemic influence* is very decided. Localities may be unvisited for several years and then exhibit a number of cases. The epidemic is seldom, however, widespread and is usually of short duration. It may be limited to a school or other public institution, or to a small portion of a city, and extend thence only slowly, lasting a few weeks or months. The varying influence of the epidemic is very marked, too, in the severity, the infectiousness, and the tendency to complications. The *individual susceptibility* is not great except where persons are closely associated, as in boarding-schools or in barracks, and the decided majority of those exposed escape. As a result the affection is much less frequent than most of the other acute infectious diseases.

Exciting Cause.—The disease is clearly an infectious one, being a blood-infection with localization usually in the salivary glands. That it is a general infection and not merely a local inflammation is proven by the cases in which the testicle is first or solely involved. The nature of the germ is still not positively known. Various studies have been made by Capitan and Charrin,⁶ Ollivier,⁷ Bordas⁸ and others, and different organisms described. Among the most important contributions is that of Laveran and Catrin⁹ who found a diplococcus chiefly in the blood or the parotid secretion in 67 out of 92 cases. These observations were confirmed by Meecray and Walsh¹⁰ and by Bein and Michaelis.¹¹ Pick¹² discovered the same organism in fluid obtained from the parotid by puncture but failed to find it in the blood. Teissier and Esmein¹³ found a micro-organism in the blood and saliva, with which they obtained a positive agglutinative reaction. Clearly, the germ is not yet certainly known. The experiments which have apparently succeeded in transmitting the

¹ Ugeskr. f. Læger 5 R. III. 5, 1896. Ref., Jahrb. f. Kinderheilk, 1898, XLVII, 313

² Revue méd. Suisse Rom., 1883, III, 81.

³ Wien med. Blätt., 1888, XI, 1613.

⁴ Brit. Med. Journ., 1902, II, 1537.

⁵ Am. Journ. Med. Sciences, 1855, XXIX, 56.

⁶ Compt. rend. soc. biol., 1881, III, 192; 358.

⁷ Rev. mens. de mal. de l'enf., 1885, III, 297.

⁸ Compt. rend. soc. biol., 1889, XLI, 644.

⁹ Compt. rend. soc. biol., 1893, XLV, 95, 528.

¹⁰ Med. Rec., 1896, I, 440.

¹¹ Verhandl. 15 Kong. f. inn. Med., 1897, 441.

¹² Wien. klin. Rundsch., 1902, XVI, 309.

¹³ Compt. rend. soc. biol., 1906, LX, 803; 853; 897.

disease to monkeys (Gordon)¹ and to cats (Wollstein)² would indicate that the virus is a filterable one.

Transmission and Period of Infectiousness.—Transmission is almost invariably direct, close proximity being required; the germ probably being contained in the breath or the saliva. It may be possible that it can occasionally be carried by the clothing of a third person, or by letters and the like, but this seems certainly uncommon. It is not transmitted by the air to any distance. The tenacity of life of the germ is probably not great. The period of greatest infectiousness is during the presence of symptoms, but numbers of instances prove that mumps can be transmitted some weeks after complete recovery; as also even before swelling appears. The mode of entrance of the germs into the system is usually from the mouth through the duct of Steno into the parotid gland. That there may be other routes is indicated by the occurrence of primary involvement of the testicle or of other salivary glands than the parotid.

Pathological Anatomy.—The benign character of the disease makes the nature of the pathological process a matter little understood, Virchow's³ opinion, based upon investigations upon secondary parotitis, was that the primary lesion is a catarrhal inflammation of the ducts with consequent obstruction, followed by inflammation of the glandular tissue and a secondary periparotitis. This latter, together with involvement of the cervical lymph-glands, accounts for the diffuse character of the swelling of the neck as the disease advances. Suppuration does not occur except through a secondary infection by pyogenic germs. This theory was supported by certain later investigators. Other views make the primary lesion an inflammation of the interacinous and periglandular connective tissue, the epithelium remaining normal.

The swelling is limited to the parotid glands in the majority of cases. Sometimes the submaxillary salivary glands are also or only affected, and the sublingual gland is less often involved. The testicles are rarely attacked in childhood.

Symptoms. Incubation.—The stage of incubation is a variable one, a general average being 2 to 3 weeks. Periods as short as 3 (Demme)⁴ and as long as 30 days (Anthony)⁵ have been reported.

Invasion.—Prodromal symptoms are absent or overlooked in the majority of cases. When present they consist of irritability, malaise, chilliness, headache, general neuralgic pain, disturbed sleep or somnolence, loss of appetite, and moderate fever. These continue from a few hours to 2 days. Sometimes earache, moderate sore throat, vomiting and diarrhea are seen. Convulsions are exceptional. Barthez and Sanné⁶ observed prodromes in 1 out of every 3 cases in their hospital practice.

Stage of Swelling.—On the 2d or 3d day of the invasion, if prodromal symptoms have been noted, evidences of local involvement appear. There is a dull, aching *pain* in the region of the ear and cheek, usually on but one side. It is made worse by pressure and often by movement of the jaws, or by the presence in the mouth of acid or cold substances. It increases gradually during several days, keeping pace with the swelling, and movement of the head may become painful. Pain in the ear is not

¹ Rep. Local Gov. Board, London, 1914. Ref., Journ. Amer. Med. Assoc., 1914, LXIII, 414.

² Jour. Amer. Med. Asso., 1918, LXXI, 639.

³ Annalen des Charité Krankenhaus, 1858, VII, 3, 1.

⁴ Wien. med. Blätt., 1888, XI, 1613.

⁵ La sem. méd., 1893, XIII, 99.

⁶ Mal. des enf., 1891, III, 696.

uncommon. The degree of pain in general varies greatly with the individual. In mild cases the mouth can be opened readily and widely, chewing offers little difficulty, and there is but little pain in the face. In severe cases the teeth can scarcely be separated, chewing is impossible, speaking and swallowing difficult, and there is much aching in the parotid gland, increased by the congestion which heating occasions.

A few hours after the first development of pain, *swelling* begins and increases rapidly. It appears first in the region of the parotid gland, the outline of which can be more or less distinctly felt below, in front of, and behind the ear (Fig. 153). Sometimes only a portion of the gland is attacked at first. The swelling spreads in every direction to an extent dependent upon the degree of involvement of the other salivary glands, the cervical lymphatic glands, and the subcutaneous tissue. In well-



FIG. 153.—MUMPS.

Swelling of left parotid gland. From a patient in the Children's Hospital of Philadelphia.



FIG. 154.—MUMPS.

Fifth day of the disease, showing eversion of the lobes of the ears, and swelling of the parotid region on both sides.

skin covering it is tense and shining, but not reddened. After the maximum is reached, in from 2 to 4 days or sometimes later, diminution

marked cases it reaches from the angle of the jaw well up toward the eye, and from the mastoid process to the anterior portion of the neck. The lobe of the ear seems lifted up and pushed outward (Fig. 154). In severe cases the process may extend about the eye, causing edema of the lid, chemosis, or even exophthalmos; or it may reach to the clavicle, or across the neck connecting the two sides. In some instances the whole side of the face is so swollen that the outline of the jaw is obliterated, and the patient may be almost unrecognizable. The swelling is hard and moderately tender on pressure. This is especially so over the central portion, where it is chiefly parotid; while in the periphery the infiltrated connective tissue offers a lesser degree of resistance. The

in the size of the swelling begins and progresses with variable rapidity.

In the great majority of cases the second parotid is attacked 1 to 2 days after the onset of the disease in the first. Sometimes the interval is decidedly longer, and the involvement of the first parotid may have entirely disappeared before that of the second begins. The degree of swelling in the two glands is frequently different. In some epidemics involvement of both parotids is much less common than in others.

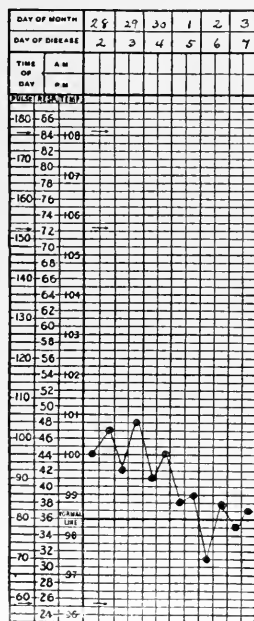


FIG. 155.—MUMPS OF MODERATE SEVERITY.

William K. F., aged 9 years. Apr. 27, swelling began in right parotid without pain or tenderness. On left side slight tenderness; Apr. 29, both sides decidedly swollen, ear-lobes everted, anything cold in mouth gives pain; May 2, swelling of right side nearly gone, that of left diminishing rapidly; May 4, right side normal, some puffiness on left side and under chin.

Quite frequently the disease attacks the submaxillary salivary glands as well as the parotids. The incidence of this occurrence varies with the epidemic. Fabre¹ found it 29 times in 58 cases, the swelling being nearly always secondary to the parotid involvement; but this frequency is rather uncommon. Nevertheless, in an exceptional epidemic reported by Spengler² the inflammation was predominantly submaxillary. Quite unusually the submaxillary glands are attacked without involvement of the parotid. This was true, for instance, in 7 of Fabre's cases, while in 9 others the infection was primary here, and secondary in the parotids. In 6 of Wertheimer's³ 77 patients the submaxillary was alone affected. In cases of submaxillary involvement, an oval, rather soft swelling is found below the jaw on one or both sides. Involvement of the sublingual gland is uncommon. It may exceptionally occur alone or be followed by parotitis (Fabre). It produces a swelling under the anterior portion of the floor of the mouth.

During the first 3 or 4 days of mumps fever continues, the degree depending upon the severity of the attack (Fig. 155). A rise to 102°F. (38.9°C.) is the average, although occasionally 104°F. (40°C.) is attained, while in mild cases it may be absent. The secretion of saliva is often much diminished, leaving the mouth dry; often unaffected or occasionally increased. Malaise and some degree of prostration are evident, the appetite poor and sleep disturbed. Swelling of the tonsils and redness of the mucous membrane of the fauces and of the mouth is not infrequent. There may be deafness and tinnitus, vomiting, diarrhea or epistaxis. In severe cases there may be headache, delirium, apathy, somnolence, and even exceptionally convulsions or the symptoms of the typhoid state with enlargement of the spleen. Very frequently, however, the children do not feel ill enough to desire to stay in bed. The urine may exhibit a

¹ Gaz. méd. de Paris., 1887, 7s, IV, 510.

² Med. Zeitung., 1885, XXI, 183.

³ Münch. med. Wochenschr., 1893, XL, 656.

febrile albuminuria if the temperature is high. Bradycardia is a common symptom, the pulse not infrequently equalling 40 or 50 in the minute (Teissier;¹ Roux).²

The *blood* in mumps, according to the investigations of Sacquepée,³ Krestnikoff,⁴ F. Pick,⁵ Wile,⁶ Feiling⁷ and others, shows slight or no absolute leucocytosis, or even a leucopenia; but always a relative increase in the number of lymphocytes and decrease of the polymorphonuclear cells.

The *duration* of the disease in average cases is 5 to 8 days, but in severer attacks it is sometimes 2 to 3 weeks before the swelling is entirely gone. With the beginning of the diminution of the swelling there is a rapid decline of the constitutional symptoms. The involvement of the second parotid some days after the appearance of the disease in the first of course prolongs the course.

Complications and Sequels.—The most important of these involve the *genitourinary apparatus*. Orchitis could with equal propriety be considered one of the forms of the disease. This is only rarely observed in subjects under 12 years of age, although in adults it is common, Comby⁸ estimating that it is seen in 1 out of every 3 cases of mumps in soldiers. Steiner⁹ recorded it in a child of 9 months, in this instance being the primary affection. Barthez and Sanné¹⁰ saw it 3 times in children of 12 years in 230 cases of mumps in early life, and Grognot¹¹ reports an instance in a boy of 2 years. It develops oftener in the 2d or 3d week of the disease, and generally only one testicle is attacked. Sometimes inflammation of the testicle is primary and occurs alone, or may be secondary to inflammation of the submaxillary glands. The pathological changes are those of a simple orchitis. The attending symptoms are in part local and in part general. The latter may be severe, consisting of high fever, prostration, feeble pulse, and sometimes vomiting, diarrhea, delirium or unconsciousness. Sometimes adynamic symptoms with low temperature are observed. After the 3d or 4th day the constitutional symptoms rapidly disappear, the resolution of the swelling taking a somewhat longer time. Recovery is generally complete, but atrophy of the gland has resulted not infrequently. Very rarely an analogous inflammation may develop in the ovaries, uterus, female external genitals or the breasts. It is possible that ovarian involvement occurs oftener than supposed, since pain and tenderness are present in a considerable number of cases, as has been pointed out by Troitzky¹² and MacNaughton.¹³ Prostatitis and urethritis are rare complications. Infectious nephritis is uncommon. J. A. Miller¹⁴ collected 30 cases from medical literature including the one reported by himself. It is oftener a sequel than a com-

¹ Bull. acad. de méd., 1912, Jan. 16. Ref. Arch. f. Kinderh., 1913, Suppl. Bd., 41.

² Thèse de Paris, 1913.

³ Arch. de méd. exper. et d'anat. path., 1902, XIV, 114.

⁴ Dissert. St. Petersb., 1902. Ref. Archiv. f. Kinderh., 1905, XLJ, 139.

⁵ Wien. med. Rundsch., 1902, XVI, 309.

⁶ Arch. of Pediat., 1903, XXIII, 669.

⁷ Lancet, 1913, II, 71.

⁸ Grancher and Comby, Traité des mal. de l'enf., 1904, I, 449.

⁹ Wien. med. Blätt., 1896, XIX, 387.

¹⁰ Mal. des enf., 1891, III, 701.

¹¹ Gaz. méd. de Nantes, 1907. Ref. Arch. de méd. des enf., 1908, XI, 279.

¹² Roussky Vrateh., 1902, I, No. 16. Ref. Nothnagel, Spec. Pathol. und Therap. 1904, Parotitis, 55.

¹³ Brooklyn Med. Journ., 1903, XVI, 115.

¹⁴ Med. News, 1905, LXXXVI, 585.

plication; is nearly always of a hemorrhagic character, analogous to the nephritis or scarlatina; and usually terminates in recovery.

Swelling of the *lachrymal glands* or of the *thyroid* or *thymus* is exceptionally seen. Leriche¹ quoted from literature 9 cases of involvement of the lachrymal glands, and Joly² reports 7 instances in 37 cases of mumps. So great a frequency is certainly unusual. Occasionally symptoms of *pancreatic* involvement are exhibited. Simonin³ observed it 10 times in 652 cases. Swelling of the *cervical lymph-glands* is a common complication, and occasionally persists as a sequel. *Stomatitis* is sometimes seen. Severe *nervous disturbances* occasionally develop as complications or sequels. Prominent here is meningitis which has been repeatedly reported. Feliciano⁴ collected 14 cases, and the subject has also been reviewed by Acker⁵ who reported 2 cases and abstracted 29 in young persons collected from medical literature. The fluid may be turbid and with an excess of cells (Chauffard and Bordin)⁶ or sero-fibrinous (Maximowitch).⁷ Paralysis of various forms may occur as a sequel. That of the face may be the result of pressure by the inflamed tissue upon the facial nerve. It may develop, too, in other parts of the body, depending upon neuritis or, rarely, meningoencephalitis. Such cerebral conditions as aphasia, choreiform states, and severe psychoses have occasionally been reported. *Involvement of the ear* has been repeatedly recorded. Deafness may be temporary or lasting, usually unilateral, depending sometimes upon an otitis media, but much oftener upon labyrinthine disease. Gallavardin⁸ and Boot⁹ each collected 51 cases of the latter. The *eye* may exhibit conjunctivitis not infrequently, and keratitis, optic neuritis, iritis, and ocular paralysis have occasionally occurred. Woodward¹⁰ collected 23 cases of optic neuritis and neuroretinitis. In a number of instances permanent blindness resulted. *Polyarthritis* dependent upon mumps has repeatedly been described. The condition has been studied especially by Lannois and Lemoine¹¹ and by Sarda¹² who saw this 7 times in 268 cases. It appears analogous to scarlatinal arthritis. *Laryngeal stenosis* is an occasional and very dangerous complication, depending upon edema or upon pressure by the swollen tissues of the neck.

In a small percentage of cases the parotid gland suppurates as a result of a secondary pyogenic infection. This is, however, infrequent in children. Gangrene is a rare sequel (Demme).¹³

Mumps may occur in combination with *other infectious diseases*, such as varicella, rubeola, pertussis, grippe and scarlet fever. Among other very exceptional complications and sequels recorded may be mentioned erythema, purpura hemorrhagica, endocarditis, pericarditis, peritonitis and pneumonia.

¹ Thèse de Paris, 1893.

² Arch. de méd. et de pharm. milit., 1903, XLI, 481.

³ Gaz. mal. inf., 1903, V, 307.

⁴ Thèse de Paris, 1907.

⁵ Amer. Jour. Dis. Child, 1913, VI, 399.

⁶ Lancet, 1904, I, 1297.

⁷ St. Petersburg. med. Wochenschr., 1880, V, 185.

⁸ Gaz. des hôp., 1898, LXXI, 1329.

⁹ Journ. Amer. Med. Assoc., 1908, LI, 1961.

¹⁰ Phys. and Surgeon, XXXIX, No. 5. Ref. Centralbl. f. inn. Med., 1908, XXIX, 67.

¹¹ Revue de méd., 1885, V, 192.

¹² Montpellier méd., 1888, X, 509; XI, 15.

¹³ Loc. cit.

Recurrence and Relapse.—Second attacks of mumps are uncommon, but occasionally seen. Some writers state that recurrence is not infrequent; but it is safe to say that the large majority of pediatricists have never observed it. Relapse occurs occasionally in the sense of a reawakening of the process in a gland just recovering or its development in the second parotid some days after the disease is completely over in the first. Barthez and Sannée¹ observed it 20 times in 230 cases after an interval of from 10 days to 3 weeks. The appearance of inflammation in the second parotid before the disease has run its course in the first cannot properly be considered a relapse.

Prognosis.—Mumps is one of the mildest and least dangerous of the acute infectious diseases; yet complications may occur and exceptionally render the attack very severe and even fatal. In Ringberg's 58,331 cases² there were but 7 deaths. Demme³ saw 2 fatal in 117 cases, both of them from gangrene of the parotid gland. Death has also occurred from a complicating meningitis or nephritis. The danger of atrophy of the testicle or of permanent deafness is to be borne in mind. In childhood, however, all severe complications are very uncommon.

Diagnosis.—This rests upon the rapid development, the characteristic situation and form of the swelling, and the course of the disease. *Acute cervical adenitis* is a frequent source of error. In it, however, the centre of the swollen area appears to be *below the jaw*, while in mumps it is just below the lobe of the ear. The course of adenitis is much more prolonged and the swelling more tender, with redness of the skin covering it. Mumps primary in the submaxillary gland cannot at first be distinguished from lymphadenitis. The more rapid course and sudden onset of the salivary inflammation, the lesser degree of induration, and the development of cases of parotid disease in the household aid in making a diagnosis. Sometimes, however, the distinction cannot be made immediately, especially if the case is not seen at the beginning of the attack. The diagnosis of sublingual involvement should be made only with great reserve, and after exclusion of other possible inflammations in the locality. Association with mumps in other salivary glands or attacking other inmates of the house would generally be necessary to make the diagnosis certain. A *secondary parotitis* occurring in the course of other diseases, such as typhoid fever, sepsis, and the like, is slower in development, unilateral, and tends to suppuration. I have more than once seen *diphtheria* supposed to be mumps, the swelling in the lymphatic glands and subcutaneous tissue of the neck being referred by the observer to the parotid gland. Only a careless failure to examine the fauces can account for the error.

Treatment. Prophylaxis.—The prevention of the spread of the disease is difficult, owing to the possibility of transmission both after the symptoms have disappeared and probably during incubation. Mild and unrecognized cases also readily spread it in schools. Quarantine to be of any service should continue 3 to 4 weeks or longer from the appearance of symptoms. Fumigation of the room is a precaution hardly required.

Treatment of the Attack.—This is purely symptomatic. The child should be confined to bed while there is fever, and given a light diet, especially one which does not require chewing. Acid substances, too, should be avoided, as they sometimes increase the pain. A mild laxative

¹ *Loc. cit.*, 705.

² *Loc. cit.*

³ *Loc. cit.*

may well be administered at the beginning of the attack, and a febrifuge should be ordered if required. Careful attention should be paid to the mouth to prevent stomatitis or to relieve pharyngitis. Pain may be alleviated by hot fomentations or by rubbing with warm olive oil, or the gland may be covered with raw cotton and the face rested against a hot water bag. A 5 per cent. guaiacol ointment has been recommended to relieve pain. Severe nervous symptoms with high fever require warm baths and other sedative and antipyretic treatment.

CHAPTER XVI

MALARIA

History.—The disease has existed since early times, and different forms of it were well described by Hippocrates. It was, however, confounded with many other affections. A clearer understanding arose after the discovery of the specific action of cinchona.

Etiology. Predisposing Causes.—*Climate* is of the greatest importance, tropical and sub-tropical countries being especially the home of the disease; where it prevails, in many localities, to an extent and with a severity unknown in cooler regions. It is, however, very widely distributed, although in temperate climates it has been steadily growing less frequent. Formerly common in the Middle and North Atlantic States, it is now generally seen but seldom, or in few regions; and in those parts of the tropics where precautions against it are taken its occurrence has been greatly lessened. *Season* is also a factor, more cases being observed in summer and autumn. All *ages* are attacked by it, but children seem especially predisposed. Even the new born not infrequently exhibit it, and cases of fetal malaria are on record in which the parasite has been found in the blood shortly after birth. The possibility of the occurrence has repeatedly been denied, and without doubt it is very exceptional; but cases which appear to be beyond question have been reported by Crandall,¹ Pies,² Lemaire, Dumolard and Laffont,³ Bass,⁴ Simms and Warwick⁵ and others; and Laffont⁶ has collected 47 observations, some of these belonging, however, to the older literature.

It should be stated that the degree of susceptibility of children is disputed. In Concetti's⁷ 360 cases occurring in the first 7 years of life, there were but 9 in the 1st year, and 24 in the 2d year. It is now, however, generally believed that it is much more frequent at an early age than these figures would indicate, and Koch's⁸ studies in Java appear to prove that infection by the parasite is very common even in infancy, especially in malarial districts. It is probable that in a large number of subjects at this period of life the symptoms are uncharacteristic and unrecognized, and that the organism may even be present without producing any clinical manifestations; and that the children later possess

¹ New York Polyclinic, 1893, I, 38.

² Monatsschr. f. Kinderh., Orig., 1910, IX, 51.

³ Bull. et mem. soc. de méd. des hôpitaux, 1910, XX, 866.

⁴ Arch. of Pediat., 1914, XXXI, 251.

⁵ Journ. Amer. Med. Assoc., 1908, LI, 916.

⁶ Thèse de Paris, 1910.

⁷ Traité des mal. de l'enf., Grancher and Comby, 1904, I, 554.

⁸ Deut. med. Woch., 1900, XXVI, 88.

a comparative immunity against the development of the disease. Epidemic influence exists in a sense, in that some years show very few cases, and other numerous ones in the same locality. The affection is endemic in some regions; rarely seen in others.

Exciting Cause.—Many later observations have confirmed the discovery by Laveran¹ in 1880 that the cause of the disease is a parasite of the sporozoa class called the plasmodium malarie, hematozoon malarie, hæmamœba, and by other names. Unlike bacteria, these germs pass through a definite life-cycle in which they exhibit forms seemingly diverse. In the process they destroy the red blood-corpuscles, producing pigment which appears in the leucocytes and in some of the tissues of the body. It was later discovered by Golgi² that there is more than one variety of the parasite, and that each produced a different form of the disease.

But a very brief description of the microorganism can be given here:

1. *The Parasite of Tertian Malarial Fever (Plasmodium vivax).*—The life-cycle of this variety in human blood is about 48 hours. The parasite consists at first of a small, hyaline, unpigmented body with lively ameboid movements, contained in the red blood-corpuscle. In 24 hours it has become about the size of the swollen, decolorized corpuscle, and exhibits a large amount of black, granular pigment. Segmentation now begins, producing a radial arrangement of the body, and in 48 hours from the beginning 15 to 20 small round spores are set free at the time of the chill, which enter other red blood-corpuscles and begin again the *asexual* cycle. Some of the mature bodies do not segment but pass through the *sexual* cycle when absorbed by the mosquito.

The Parasite of Quartan Malarial Fever (Plasmodium malarie).—The life-cycle of this form is 72 hours. It appears at first as a small, hyaline, unpigmented body with very little and very slow ameboid movement. In 48 hours it grows to about $\frac{1}{3}$ or $\frac{2}{3}$ the size of the red blood-corpuscle containing it, becomes more pigmented, and loses all movement. In 60 hours it fills the red blood-cell, which is not enlarged or decolorized. Segmentation now begins, producing a star-shaped or "daisy"-shaped body. This is followed by sporulation at 72 hours at the time of the chill, 5 to 10 spores being discharged to enter other red blood-cells. Certain mature bodies do not segment, as in the case of the tertian parasite.

3. *The Parasite of Estivo-autumnal (Tropical) Fever (Plasmodium precox).*—The life-cycle of this parasite is probably variable, ranging from 24 to 48 hours. It consists at first of a very small body, hyaline or with little pigment, actively ameboid, or, when at rest, having a ring-form. It develops pigment which is distributed about the periphery of the organism. It is much smaller than the preceding forms, and the red blood-cell containing it is much shrunken. The later stages of development take place in the internal organs, especially the spleen and the bone marrow, where the concentration of pigment, segmentation, and sporulation occur. After the attack has lasted about a week crescentic pigmented bodies appear free in the blood serum. These are the forms of the parasite which later pass through the sexual cycle in the body of the mosquito. They are characteristic of estivo-autumnal malaria. In the course of development some of them form flagella.

Transmission.—Malaria, although an infectious disease, is not contagious in the ordinary sense; *i.e.* it is impossible for one individual to

¹ Bull. de l'acad. de méd., 1880, XLV, 1235.

² Gaz. degli osp., 1886, VII, 419.

contract it directly from another, except by experimental inoculation with the blood of the patient. That it may be given in this way was first demonstrated by Gerhardt¹ and since then repeatedly by others. Manson² was the first to indicate clearly that the disease was communicated to man by the mosquito, which acted as host; Ross³ showed that the parasite developed in the body of mosquitoes which had bitten malarial patients, and Grassi and Bignani⁴ succeeded in transmitting the disease directly from the insect to man. Various species of the genus *Anopheles* are the only ones harmful. The insects acquire the parasite, as far as known, only by sucking the blood of a patient with malaria. In their bodies the organism then passes through another, viz. a sexual, cycle of development different from that seen in man. The *duration of life* of the plasmodium is uncertain, since whereas the asexual, segmented form is present in human blood only during the attack of malaria, the sexual, non-segmented form may persist there an indefinite time without producing symptoms. This accounts for the occurrence of relapses after intervals of months. It explains, too the multiplication of cases at the season of the year when anopheles begin to be abundant, since the insects then acquire the parasites from such individuals.

Pathological Anatomy.—Many of the changes found depend upon the extensive destruction of the blood which the parasites produce. The spleen is always more or less enlarged; very soft, especially in children; and exhibits pigment and broken-down corpuscles. The enlargement in chronic cases is great. The liver may be hypertrophied and contain pigment, which gives it a slate-brown color; the kidneys enlarged and gray-red, with pigmentation of the glomeruli. The lesions of acute nephritis are sometimes seen, or chronic nephritis after repeated attacks of the disease. The gastrointestinal mucous membrane and even the skin may exhibit pigment.

Symptoms. Period of Incubation.—The duration of this period is uncertain. Even in the disease produced experimentally it varies from 3 to 21 days (Mannaberg).⁵

Typical Forms.—The ordinary forms of the disease occur in adults and in later childhood much oftener than at earlier periods. Prodromal symptoms are often present, lasting 1 or more days and consisting of malaise, loss of appetite, vertigo, chilliness, yawning, and irritability. A distinct paroxysm then develops, marked by headache, lassitude or prostration, coated tongue, general pains, and often vomiting. There promptly follows an evident chill of varying intensity lasting 10 minutes to an hour or more, with shaking of the body, chattering of the teeth, sensations of cold, and a pinched and blue appearance of the face. In this, the *cold stage*, although the surface of the body feels cold to the touch, the thermometer shows a decided rise of axillary or rectal temperature. The pulse is small. The second, or *hot stage*, is marked by the beginning of the sensation of fever. The temperature reaches 104° to 106° F. (40° to 41.1°C.), the skin is flushed, the pulse full and accelerated, and headache and thirst are complained of. The spleen can often be felt. The maximum temperature as shown by the thermometer is reached during or shortly after the chill. The duration of this stage is from 3 or 4 up to 12 hours.

¹ Zeitschr. f. klin. Med., 1884, VII, 372.

² Brit. Med. Journ., 1894, II, 1306.

³ Brit. Med. Journ., 1897, II, 1786.

⁴ Ref. Mannaberg, Nothnagel's Encycl. Pract. Med. Amer. Edit., Malaria 117.

⁵ Nothnagel's Encyclop. Pract. Med., American Edit., Malaria, 103.

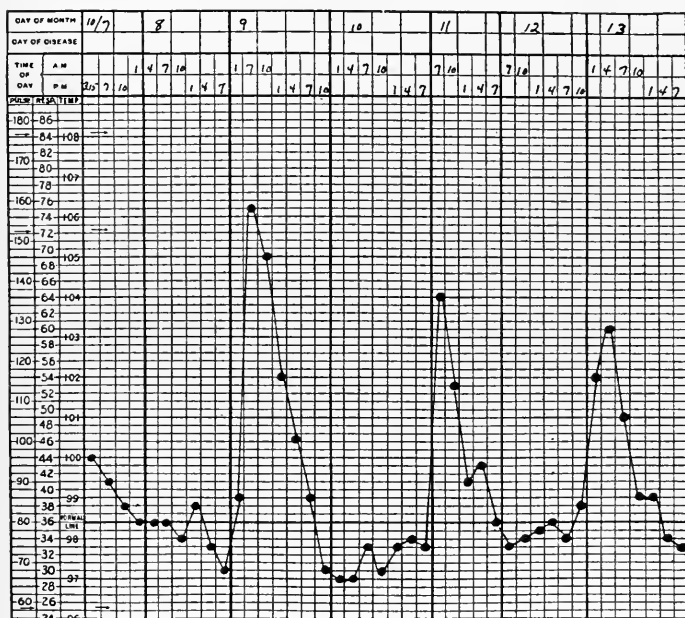


FIG. 156.—MALARIA, SIMPLE TERTIAN.

John T., aged 2 years. Oct. 6, fever, thirst, diarrhea; Oct. 7, spleen large, free urination; Oct. 9, plasmodium found in blood; Oct. 13, paroxysm anticipated several hours.

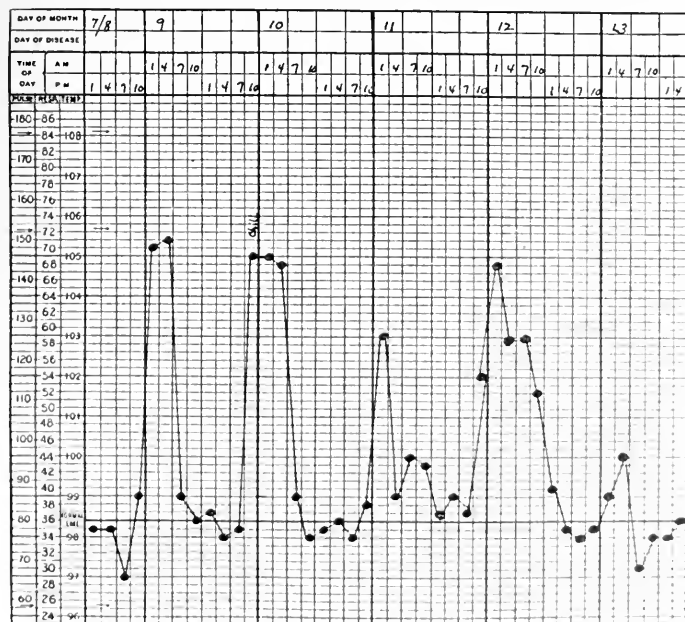


FIG. 157.—MALARIA, DOUBLE TERTIAN.

Lizzie Q., aged 8 years. Been having chill every night followed by fever. Vomited once. Examination showed enlarged spleen and parasites in blood. Attacks controlled by 20 grains (1.296) of quinine by mouth and 10 grains (.648) by suppository given on 12th. Previous small doses without effect.

The second stage of the paroxysms is gradually replaced by the third, or *sweating stage*, in which all the uncomfortable symptoms of fever disappear and the temperature rapidly returns to normal. The amount of sweating is very variable. Comfortable sleep usually follows. The total duration of the paroxysm averages 6 to 12 hours. After the paroxysm the patient usually feels entirely well until the next one occurs, the length of the interval depending upon the type of malaria present.

The urine in malaria frequently exhibits albumin in moderate amount. It may often be scanty just before the chill and increase during and after the paroxysm. The blood exhibits besides the parasites, a very decided and rapidly developing anemia in cases which have had several paroxysms.

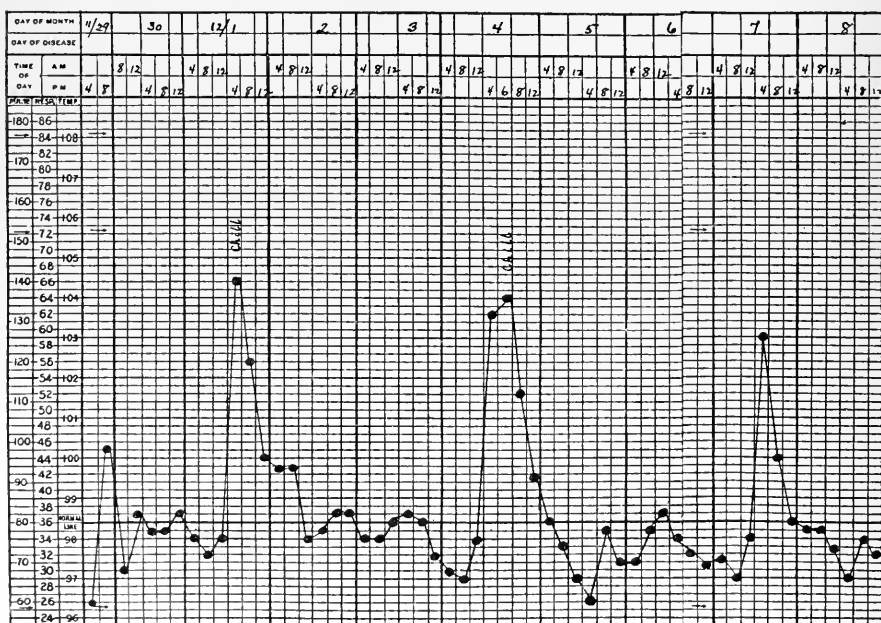


FIG. 158.—MALARIA, QUARTAN.

Lawrence G., aged 16 years. Tertian fever a year before. Chills at intervals for over a year. Blood showed typical quartan bodies. Spleen much enlarged. (*Thayer & Hewettson's "Malarial Fevers of Baltimore."*)

Leucocytosis is rare in malaria, but a relative increase of the large mononuclear cells is considered characteristic by some investigators. The leucocytes often contain pigment-granules.

Instead of beginning at almost the same hour on the days of the attacks, which is the rule in malaria, the paroxysms sometimes "anticipate" slightly, occurring from 1 to several hours before the full period has elapsed (Fig. 156.) Under treatment with quinine "postponement" of the paroxysms for some hours is sometimes observed.

The description of the symptoms as given applies to the intermittent form of the disease and as occurring in older children. In many instances, however, especially in tropical climates or in young children anywhere, malaria assumes an irregular, continuous, or remittent type, with the various stages of the paroxysm less marked, or not at all so.

Varieties of the Typical Form.—1. *Tertian Malarial fever*, the most frequent form in temperate zones, is that produced by the tertian parasite. In *Simple Tertian* but one set of germs is present, and paroxysms occur every other day, usually at about the same hour (Fig. 156). Should, however, two sets of tertian organism be present, reaching the stage of sporulation on alternate days, *Double Tertian*, or *Quotidian* fever occurs (Fig. 157), the patient having a paroxysm every day.

2. *Quartan fever*, depending upon the quartan parasite, exhibits a paroxysm every 4th day; *i.e.* with free intervals of 2 days (Fig. 158). Should two sets of the organism be present, paroxysms occur on two con-

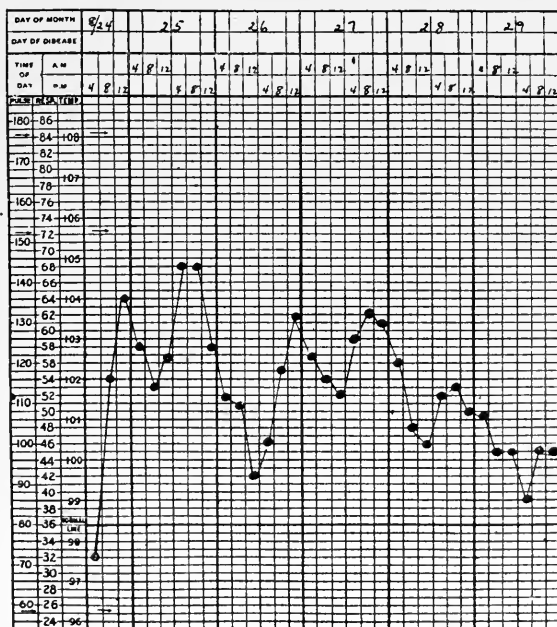


FIG. 159.—MALARIA, AESTIVO-AUTUMNAL. SHOWING IRREGULARLY INTERMITTENT TEMPERATURE.

Emma B., aged 13 years. Illness began 10 days before, pain in abdomen and side, cough, enlarged spleen, crescentic bodies in blood. No chills or distinct paroxysms. Quinine treatment commenced on 28th. (*Thayer & Hewettson's "Malarial Fevers of Baltimore."*)

secutive days, with one day free. If three sets are present, quotidian (*i.e.* triple quartan) fever is produced.

3. *Estivo-autumnal Fever, or Tropical Fever.*—Both the other forms described are often denominated "*Intermittent fever*," since the temperature is normal for at least a part of every day. In the form due to the estivo-autumnal parasite the attack is usually of a *remittent* type; *i.e.* the temperature, although lessening at intervals, does not reach normal, and the paroxysms, if present, are of longer duration (Fig. 159). Jaundice is common and gastrointestinal symptoms may be marked. In other cases the individual paroxysms may be little or not at all observed, and great irregularities in the course are liable to be exhibited, and in still others the temperature is continuous, with little variation, and the case may resemble typhoid fever closely. Sometimes, although

dependent upon the estivo-autumnal parasite, the attack is distinctly intermittent and either quotidian or tertian in character. The cold stage is, however, absent or less marked, the rise and fall of temperature are usually more prolonged, and the patient does not feel so well in the apyretic period as when the disease is due to the tertian parasite. The cases show, also, an obstinate tendency to relapse.

Cases of estivo-autumnal fever, occur in both temperate and tropical

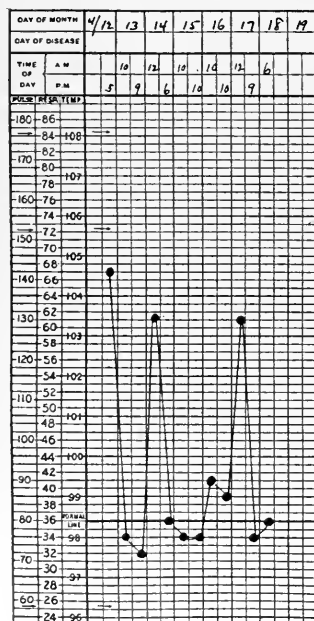


FIG. 160.—MALARIA, TERTIAN TYPE SHOWING IRREGULARITY, WITH ABSENCE OF RISE OF TEMPERATURE ON ONE OCCASION.

Mamie R., aged 2 years, 8 months. Cough and cold in head 2 weeks previously, lost appetite. A week before seen began to have fever every other day. Headache, pain in abdomen, drowsy. Examination on admission to the Hospital of the University of Pennsylvania showed spleen slightly enlarged to palpation. Nothing else of note. Malarial organisms found Apr. 19. Child removed. Quinine not administered.

The typical course is more often absent than present at this time of life. The onset is more abrupt, the whole paroxysm is often shorter, and the division of the attack into stages is frequently absent or little marked. In infants especially the symptoms may be so irregular that the disease is frequently overlooked. The chill in early life is usually replaced by mere coldness, pallor, and blueness of the face and extremities; or by yawning and drowsiness, or not infrequently convulsions. Vomiting is

regions, but those of the severer form are encountered principally in tropical and sub-tropical countries, including the Southern United States. They may pass into the *pernicious type*. The *algid form* belongs here. It is marked especially by an extreme sensation of coldness, scanty urine, excessive prostration, vomiting, and sometimes profuse diarrhea. The temperature is, at most, only slightly elevated; often sub-normal. Respiration is accelerated, the pulse weak, and the mind clear. Another variety of pernicious malaria is the *comatose form*, with high fever and rapidly developing unconsciousness and delirium. The *hemorrhagic form*, or "blackwater fever," is that variety of pernicious malaria characterized especially by hemoglobinuria, jaundice and uncontrollable vomiting. It is very uncommon in childhood. It is to be noted that occasionally cases of the pernicious type may be produced by the tertian or quartan parasite.

Tertian malarial fever is by far the most frequent form of the disease in children in temperate zones, the quotidian type being especially common. Quartan fever is rare in the northern portions of the United States, and in the Southern States is less frequent than other forms. The estivo-autumnal fever is the prevailing form in tropical regions. Occasionally there is a combination of the estivo-autumnal type with the other varieties.

IRREGULAR FORMS.—It is particularly in infancy and early childhood, although by no means solely then, that variations from the definite types of the disease are seen, or that certain symptoms are especially prominent.

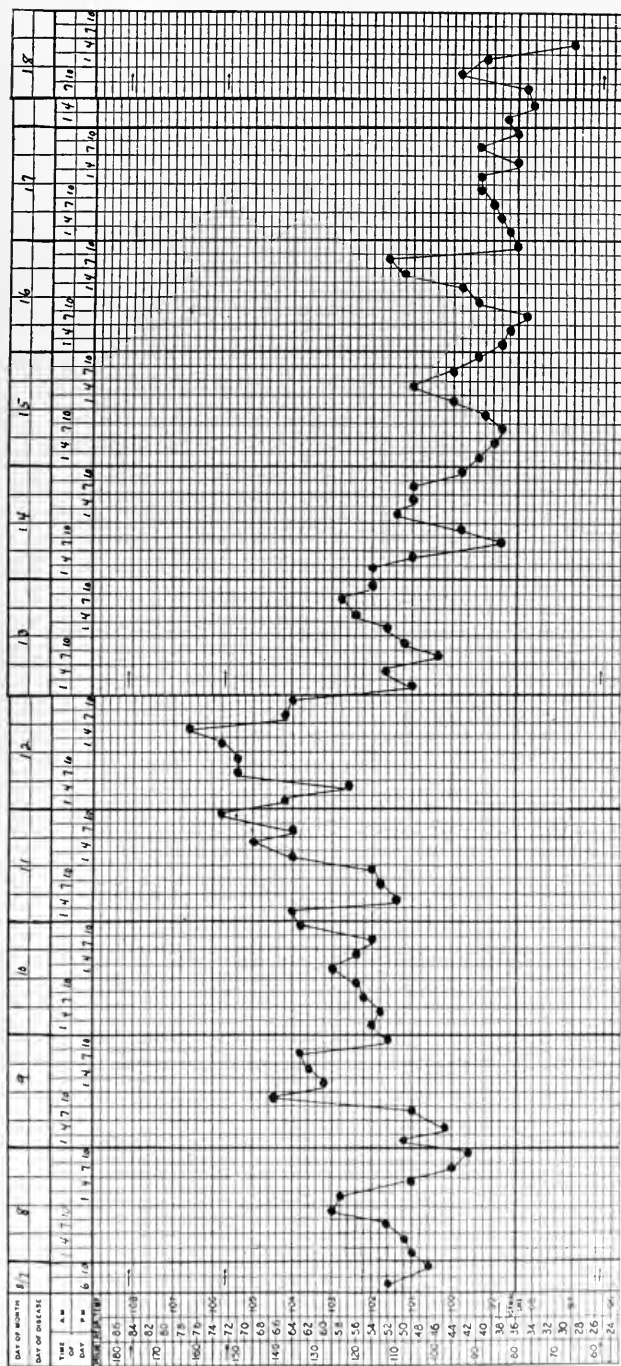


FIG. 161.—MALARIA, IRREGULAR INFANTILE TYPE.

Harriet L., aged 19 months. Been ill for 5 days with loss of appetite, malaise, diarrhea and repeated vomiting. Examination showed enlarged spleen and liver and numerous plasmodia in the blood. Given 10 grains (.648) of quinine daily, by suppository, beginning on Aug. 9, without effect; and 10 grains (.648) quinine, taken by mouth, daily, beginning on the 13th.

a common initial symptom. In the hot stage vomiting may continue and there is coating of the tongue and loss of appetite. Constipation may be present, but diarrhea is very common and sometimes is profuse. The younger the child the more liable is diarrhea to occur. The infant exhibits either drowsiness, or restlessness and crying. The temperature is usually higher in early life and is prone to run a much more irregular course, approaching sometimes, even in tertian fever, a remittent or continuous type (Figs. 160 and 161), due, probably, to the presence of a number of sets of the tertian parasite. Nervous symptoms are prominent. Headache and pain in the epigastrium, limbs, or splenic or hepatic region are very frequent and delirium is not uncommon. Bronchitis is a very common symptom and not infrequently there may be a degree of pulmonary congestion, especially in infancy, which suggests pneumonia. The sweating stage is absent or little marked in children under 2 years of age, and when seen is liable to be attended by considerable prostration.

Latent Malaria.—In some cases malaria may be "latent," the usual symptoms, including fever, being absent, and the disease suggested only by such obscure manifestations as a periodically recurring cephalalgia or cardialgia, periodic diarrhea, periodic torticollis, or an obstinate cough which yields only to quinine. It is to be noted, too, that the malarial parasite may remain dormant in the system for months, and probably indefinitely, without producing symptoms, the patient having at times recurrent attacks.

Chronic Malaria. Malarial Cachexia.—In patients who have had repeated attacks of malaria a cachexia develops, characterized especially by large spleen and great anemia, with consequent debility, pallor, and dropsy of the skin and sometimes of the serous cavities. There is usually little or no fever, or slight rises occur only for a short period in the 24 hours. The general symptoms are indefinite, consisting of anorexia, coating of the tongue, malaise, debility, cough and vague pains. There is, in fact, nothing especially suggestive, unless the patient is a resident of a malarial district, and only the examination of the blood can determine the diagnosis.

Complications and Sequels.—One of the most prominent complications is bronchitis. As already pointed out, it may sometimes be the most suggestive evidence of the disease. Congestion of the lungs often occurs and even exceptionally a fully developed pneumonia. Epistaxis is an occasional complication. Gastrointestinal affections of moderate severity are sufficiently frequent to constitute a symptom of the disease. Vomiting, however, may become very obstinate and diarrhea may at times take on a dysenteric or choleraic character. Jaundice is not uncommon. Herpes is seen very frequently and urticaria and erythema occasionally, and symmetrical gangrene has been reported. Endocarditis and torticollis have been observed and neuritis is an occasional sequel. Aphasia and hemiplegia or other paralyses have been observed. Acute glomerular nephritis occurs not infrequently in severe cases and chronic nephritis may develop in malarial cachexia. Glycosuria has been frequently observed and vesical irritability with enuresis is sometimes seen. Malaria may occur in combination with or as a sequel to other infectious diseases as, for instance, pertussis, scarlet fever, smallpox, and syphilis. In some regions it is frequently combined with typhoid fever.

Recurrence and Relapse.—The tendency to relapse is one of the great characteristics of malaria. The germs may lie dormant in the

system for an indefinite period and the disease reappear at any time. How often the new attack is a true *relapse* due to an infection already present, and how often a *recurrence* through a reinfection from without, it is usually impossible to say, unless the patient has removed to a locality certainly free from anopheles. The occurrence of one attack usually seems to render the subject particularly liable to later ones. On the other hand, the studies of Koch¹ have confirmed the opinion of many physicians in the tropics that infected individuals may finally become immune, and that the natives of some malarial districts do not readily develop the disease because of having had an attack in infancy.

Course and Prognosis.—The duration of the disease is uncertain. Unless treated with quinine it may continue indefinitely and pass into the chronic stage. In other cases, particularly of the tertian type, it stops of itself after a week or more, although very prone to relapse frequently. The pernicious cases end fatally in a day or two. Malarial cachexia may last months or years before complete recovery is obtained. The prognosis varies with the locality. In malarial regions, especially in the tropics, the disease reaches the proportions of a plague, and is the cause of many deaths. In temperate climates the prognosis is usually good in districts where the disease is sporadic or occurs only exceptionally in epidemic form. This is most true of the intermittent type, which tends to recover spontaneously, particularly if the subject is removed from malarial districts and repeated reinfection prevented. The irregular forms run an indefinite course, and when they are dependent upon the estivo-autumnal parasite they are liable to be severe. In the Southern States of this country and in tropical regions the estivo-autumnal form is attended by a higher mortality in infancy, and death may take place in a few hours during the first attack. This applies, however, only to the pernicious variety of the disease. Under treatment and outside of malarial districts the prognosis of malaria is favorable, and death rarely occurs except from complications. The great tendency to relapse has already been referred to.

Diagnosis.—The diagnosis is easy in typical cases, but often presents great difficulties in the anomalous and irregular forms, which are especially liable to develop in early childhood and infancy. A certain tendency to periodicity may sometimes be discovered in these cases, which is very suggestive, as is also enlargement of the spleen; but the most satisfactory diagnostic characteristics are the cure by quinine, and the discovery of the parasite in the blood. This latter can usually be accomplished when quinine has not been administered for any length of time, but considerable practice and experience are required. Consequently the failure to find the germs is no proof that the disease is not malaria. A single negative result of examination should not suffice, inasmuch as the organisms may at the time be absent from the peripheral circulation. The study is best made a few hours before the paroxysm is expected. At its height they may not be discoverable. On the other hand, a fever of intermittent type which is not cured by quinine is almost certainly not malaria, and even the more irregular and remittent types will generally respond to this therapeutic test, if sufficiently large doses are administered. It must be remembered, however, that the cure, although usually prompt, is not invariably so.

There are a number of disorders which simulate malaria closely, prominent among them being *suppurative conditions*, such as empyema,

¹ *Loc. cit.*, 88.

pyelitis, and septic processes of any sort. These may exhibit an intermittent fever with chills and sweats. Careful local examination may be required to show their true nature. The failure of quinine to relieve them permanently, and the presence of leucocytosis, show that they are not malaria even without an examination of the blood for the parasites being made. Malarial fever may also be closely simulated at times by *tuberculosis* and by *typhoid fever*. The latter, especially, may be very like the remittent form due to the estivo-autumnal parasite, and sometimes, too, strongly suggests the intermittent type of malaria. (See Typhoid Fever, p. 399, Fig. 99.)

Rickets, *syphilis* and forms of *splenomegaly* may exhibit both anemia and enlargement of the spleen, but have nothing else to suggest the malaria cachexia.

It was a habit, very common in former years of attributing to malaria numerous obscure conditions for which no satisfactory explanation could be found. This has been the cause of many a fatal error in diagnosis. No doubtful case should be called malaria unless an examination of the blood or successful treatment with quinine shows that it really belongs in this category.

Treatment. Prophylaxis.—This consists in the destruction of the anopheles, the protection from the bites of the insects, and the prevention of relapse in those who have had the disease. The first of these has been successfully undertaken on a large scale by governments in some regions. All accumulations of water or other breeding places should be sprayed with kerosene. The avoidance of relapse is to be accomplished by the frequent administration of small doses of quinine, especially at the time of the year when malaria is most liable to occur. Individuals who are obliged to visit a malarial region should take quinine in small doses constantly. Mosquitoes should be kept as far as possible from biting by the use of mosquito-nettings at night; by seeing that the children are in the house before sunset, since the anopheles do their biting at night; and by applications to the skin of an ointment containing pennyroyal, menthol, tar, citronella or other substance disagreeable to the insects.

Treatment of the Attack.—Quinine is a specific, and the only one, although good results have been reported from arsenic in large doses. To subjects old enough it can best be given in capsules; for those younger it must be in solution. Either the sulphate (74.31 per cent. quinine), the bisulphate (59.12 per cent.) the hydrochloride (81.71 per cent.) or the dihydrochloride (81.61 per cent.) may be employed. For solutions the last is the best, as it is very soluble and is fairly rich in the quinine base. The sulphate is comparatively insoluble. The taste of these salts may be disguised to a certain extent by the admixture of syrup of yerba santa, syrup of licorice, syrup of chocolate, or other pleasant menstruum. The tannate (30 to 35 per cent. quinine) is much less effective, but in the form of "quinine chocolates," has very little unpleasant taste and is often taken readily by children. Euchinin (84 per cent. quinine) and aristochin (96 per cent.) are two comparatively tasteless quinine derivatives which may be used instead of those mentioned.

Quinine should be given, when possible, in a single large dose 2 or 3 hours before the time of sporulation of the parasites, which coincides with the occurrence of the paroxysm, since at this time the germs are outside of the red blood-cells and are more easily killed. A great objection to this method is the tendency to cause vomiting. When this occurs it is better to administer the drug in smaller doses every 2 or 3 hours

beginning 3 or 4 hours after the paroxysm is over and giving, if possible, a slightly larger amount shortly before the time of the expected recurrence.

In the irregular or remittent forms, especially in serious cases, the patient should receive a large dose as quickly as possible, and then be kept fully under the influence of the drug by smaller doses repeated at regular intervals. A somewhat larger amount is generally required in such cases, particularly if the organism is of the estivo-autumnal type. Sometimes quinine is borne better immediately after food, sometimes on an empty stomach. When vomiting cannot be overcome, and in all pernicious cases, the remedy must be given by the bowel, either by enema or suppository, or hypodermically. For hypodermic use the dihydrochloride of quinine, or the hydrochloride of quinine and urea, may be employed. The hypodermic employment of quinine is, however, liable to be very irritating, and may be followed by abscess. It is to be selected only when other methods fail, and the drug should be given in a few large doses rather than in repeated smaller ones. After the attack of malaria has been controlled quinine should be continued in somewhat smaller doses for 2 or 3 weeks, or relapse will be very liable to occur.

Dose.—Quinine is borne by children in relatively large doses, and, except for the tendency to produce vomiting, appears to be a safe remedy. Taking the sulphate as the type, and selecting that one of the salts to be used with due regard to its comparative basic strength, an infant of 1 year, with a mild attack of malaria, should receive 6 to 8 grains (0.39 to 0.52) in the course of 24 hours, and sometimes much larger amounts are needed. In later childhood the dose may be as large as for adults. The dose for hypodermic use for 24 hours should be about the same as for administration by the mouth. For rectal employment it should be 2 or 3 times as large, since absorption is not perfect.

Treatment of the Paroxysm.—Little is needed as a rule, and that is purely symptomatic. Warm covers and dry heat may be employed in the cold stage, and sponging, if necessary, in the hot stage.

Treatment of Chronic Malaria.—Not only is quinine needed here, but often such tonics as iron, arsenic, and the like, to overcome the anemia. Removal from a malarial region is all-important.

CHAPTER XVII

TETANUS

(Lock Jaw)

History.—The disease was known to the ancients and was described by Hippocrates. It occasionally attacks older children, the cause and symptoms being the same as in the case of adults; while in the new born it is sufficiently common and important to receive in many textbooks a separate description under the title of Tetanus Neonatorum.

Etiology. Predisposing Causes.—One of the most important is that of *age*, the great majority of cases in infancy occurring in the new born. The influence of age is, however, greatly modified by locality. In some countries tetanus neonatorum has at times been endemic, and the cause of many deaths. These regions are often widely separated and of entirely different climatic conditions. Thus, it has in certain periods been very frequent in the Hebrides, in parts of the West Indies and of the South-

ern United States, and in a portion of Long Island. Of 23,398 infants dying in Roumania in the 1st month of life according to Miron,¹ 10,257 were cases of tetanus. In general it is most frequent in hot climates. The disposition of the disease depends, however, not so much on climatic influences as upon the *absence of cleanliness*. Under proper hygienic conditions it is rare. The fact that the Negro infants in some regions of the Southern States are especially liable to it is not a proof of any special predisposing influence of race, but rather of the greater carelessness regarding hygiene common in these localities among the Negroes.

The *presence of a wound*, although minute and perhaps undiscovered, is apparently necessary to the development of the disease. From this standpoint all cases of tetanus must be considered as traumatic. Punctured, lacerated and contused wounds, especially of the hands and face, are those particularly dangerous; clean, incised wounds much less so. Consequently, cases frequently develop after Fourth of July accidents, or those associated with similar celebrations. I have seen it, for instance, follow a punctured wound by a nail in the ruins of a stable, this illustrating the connection of the disease with the horse. It is very probable that abraded surfaces of the mucous membrane of the intestine offer a portal of entry in some instances. The wound of ritual circumcision or that of vaccination, if imperfectly cared for, may readily give rise to the disease. (See Vaccination, p. 382.) Much the most frequent portal of entry in the new born is the umbilical wound.

Exciting Cause.—The direct cause has been proven to be the *bacillus tetani*, discovered by Nicolaier in 1884² and isolated by Kitasato in 1889.³ This is found in garden soil and in the dust of the streets in nearly all countries, although particularly abundant in some localities. Derived from this source it is found, too, in the intestinal canal of some herbivora. It is chiefly on account of the occurrence of the disease in cattle that the occasional danger from vaccine virus arises when proper precautions have not been taken in its production. (See Vaccination.)

The bacilli inhabit the superficial portion of the wound of the patient, where an exceedingly virulent poison, the tetanus-toxin, is produced and whence very rapid absorption of this takes place. The poison exerts a special action on the motor cells in the medulla and on the anterior horns of the spinal cord. Nearly all investigators agree that the germs do not enter the blood or the organs. The disease is readily inoculated into animals. The spores of the bacilli are very tenacious of life, are uninjured by exposure to air or light, and offer considerable resistance to the temperature of boiling water. The action of disinfectant solutions also must be prolonged to accomplish their destruction.

Pathological Anatomy.—There are no characteristic post-mortem lesions. Congestion of the brain and spinal cord and of their membranes occurs frequently, but is probably the result of the convulsive condition rather than of the poison. The lungs are usually congested likewise. There is nothing peculiar about the condition of the wound.

Symptoms.—These vary slightly according as the disease affects the new born or older children.

Tetanus Neonatorum.—The *incubation* varies from a few hours to 10 or 12 days or, exceptionally, longer. In the majority of cases symptoms

¹ II Cong. d. Ruman, Gessellsch. f. d. Fortsch. u. Verbreit. d. Wissensch., 1903, Sept. 22. Ref. Schmidt's Jahrbücher, 1904, B. CCLXXI, 206.

² Deutsch. med. Wochenschr., 1884, X, 842.

³ Zeitschr. f. Hyg., 1889, VII, 225.

appear toward the end of the 1st week of life. The *onset* is marked by restlessness, crying, sleeplessness and difficulty in nursing, the infant grasping the nipple and pressing it between the jaws and then dropping it with a cry of pain. On examination the masseters will be found contracted and hard and the jaws can be forced open only with difficulty (*trismus*). The eyes are closed, the forehead wrinkled and the lips pouting. In a few hours the stiffness extends to the muscles of the trunk and limbs, at times attended by arching of the body. To these symptoms are next added violent increase of the tonic contraction; this coming on in paroxysms which last a few moments, and which are repeated in severe cases perhaps every few minutes, but in milder cases at much longer intervals. They are brought on by such slight causes as movement of the body, a draught of air, and the like, or occur without any discoverable reason whatever. During well-developed paroxysms the surface of the body, especially the face, becomes red, cyanotic and swollen; the eyes injected; and the labial commissure pulled downward and outward, producing the *risus sardonicus*. (See Fig. 113, under Cerebrospinal Fever, p. 421.) The jaws are closed and foam comes from the lips; the head is retracted; the spine often much arched; the arms and thighs extended; the forearms and legs extended or slightly flexed; the thumbs bent into the palms; the fingers clinched; the toes flexed; and the muscles of the chest and abdomen hard. The whole body is as rigid as a rod of iron. Sometimes the infant is supported only on its heels and the back of its head. The respiration is irregular, superficial and difficult; swallowing impossible; the pulse rapid and weak, and the cry feeble. Jaundice is not uncommon. Slight clonic spasmodic movements sometimes accompany the attack.

The persistence of tonic contraction, especially of the *trismus*, appears to vary greatly with the case. In the severest it is practically unbroken almost from the beginning; in the milder ones complete relaxation occurs for quite long intervals. The temperature of tetanus is variable and often irregular; moderate fever or even normal temperature being the rule in the milder cases; higher fever and occasionally very high final temperature in the severer ones. Sometimes the low temperature of collapse develops.

Tetanus in Older Infants and in Childhood.—The symptoms differ in no way from those seen in adults. After a very variable *incubation* period, usually not over 10 days from the reception of a wound, *symptoms* appear, the patient complaining of chilliness, headache, fever, stiffness in the neck, and especially of difficulty in masticating and in opening the mouth. Gradual increase of stiffness in the muscles of the jaws develops, making it impossible for the patient to separate them; and the *risus sardonicus* appears. The stiffness then extends to the muscles of the body in general, especially those of the back. Distinct paroxysms now occur, similar to those described under tetanus neonatorum, the body being bathed in perspiration; the pain very intense, and the body often in the position of opisthotonos, but sometimes in a straight position (orthotonos) or drawn to one side (pleurothotonos); or doubled forward by the contraction of the abdominal muscles (emprosthotonos). There may be partial relaxation between the paroxysms. Sometimes only certain regions are attacked, especially the head and neck. The condition of the mind is normal throughout; the eyes are usually not involved. In the acute, rapid cases the paroxysms are very frequent and the fever high; in the more chronic ones incubation is longer, the periods of relaxa-

tion between the paroxysms more prolonged and complete; and the duration of the attack greater.

Course and Prognosis.—The *duration* of fatal cases of tetanus is seldom more than 3 to 4 days; sometimes less than 24 hours or as long as 6 days. In cases which recover the attack may last several weeks, the paroxysms gradually diminishing in number and severity. The more severe the case, the more rapid is the course, and the more frequent the paroxysms. Death is the result of exhaustion, collapse, or of interference with respiration.

The *prognosis* of tetanus neonatorum is extremely bad. Death generally occurs. Probably the most favorable statistics give a mortality of nearly 50 per cent., and the usual death-rate is decidedly greater than this. Only the cases of long incubation and slow development of symptoms offer much hope from treatment. The mortality appears to be inversely proportional to the length of incubation. When under 5 days it is extremely unfavorable; decidedly more favorable when over 10 days.

In tetanus after the period of the new born the prognosis is somewhat better, although still very grave. The absence of fever and the slow and late development of symptoms are the most favorable indications, the prognosis being very much better when incubation is over 10 days. The average death-rate varies from 50 to 80 per cent. Involvement of respiration is very unfavorable.

Diagnosis.—In well-marked cases the diagnosis is easy. The disease is closely simulated by the convulsions of *strychnine poisoning*, except that there is in this condition complete relaxation between the paroxysms, and that trismus rarely occurs, and certainly never early. Severe cases of *tetany* sometimes simulate tetanus, and I have more than once seen errors in diagnosis between the two diseases. In tetany, however, the muscles of the limbs are primarily and chiefly affected, and involvement of the jaws and of the neck seldom or never occurs. In cases of intermittent tetany the periods of relaxation are much longer and more complete than in tetanus. The peculiar electrical reactions of tetany are absent in tetanus. The history of the ailment is often serviceable in arriving at a conclusion. I have seen *meningitis* in rare instances strongly suggest tetanus. There is, however, in this the absence of trismus, and the presence of tenderness on moving the neck and of a disordered mental state.

Treatment. Prophylaxis.—Most careful antisepsis of the umbilical wound is the greatest safe-guard against tetanus in the new born. In regions where the disease prevails, consideration must be given, too, to the character of the water employed for the first washing of the child, as well as the cleanliness of the towels, and the like. When a case of tetanus has occurred in a lying-in institution careful disinfection of the room is to be carried out, the infant isolated, and all other exposed infants given an immunizing dose of tetanus antitoxin.

Treatment of the Attack.—The first indication is antiseptic treatment of the wound, in order to prevent further production of the poison. The patient should be handled very little and kept as quiet as possible in a darkened room, in order to diminish the tendency to paroxysms. Drugs to quiet the nervous system should be given in frequently repeated doses, large enough to produce some physiological effect. Chloral is one of the best for this purpose. A child of 2 years may begin with 2 or 3 grains (0.13 to 0.19) every hour, the amount to be increased if necessary. Potassium bromide is another valuable remedy, the dose at 2 years being 8 to

10 grains (0.52 to 0.65), or eventually more, every 2 hours. When swallowing is impossible, remedies may be administered by the bowel or through a nasal tube. Physostigma has long been a favorite remedy. Its alkaloid, eserine, may be given hypodermically in doses of $\frac{1}{500}$ of a grain (0.00013) at this age, repeated as needed. It is to be borne in mind that both chloral and eserine are depressant remedies. This statement merely indicates the necessity of care in their employment, not avoidance of them. Morphine hypodermically may be useful. The administration of an anesthetic may be necessary during the paroxysms if they are severe. Feeding is important and may be accomplished by the rectal or nasal tube if the ability to swallow has ceased.

In recent years the value of magnesium sulphate for the control of the spasms appears to have been thoroughly demonstrated. It should be administered subcutaneously, and in severe cases intraspinally or intravenously. For subcutaneous injection the dose should be 0.6 to 0.8 c.c. (9 to 13 m.) of a 25 per cent. solution per kilogram of body weight, 3 or 4 times a day (Meltzer).¹ For intraspinal and intravenous administration the solution should be weaker and the dosage much smaller. A survey of the reported cases in which the treatment has been tried is given by Robertson.² The treatment is not without danger of producing respiratory paralysis.

Antitoxin Treatment.—In 1890 Behring and Kitasato³ were able to immunize rabbits against, or to cure them of, tetanus, by injecting a tetanus antitoxin, and soon afterward antitetanic serum was employed in man and has been used repeatedly since that time. The results on the whole have been unsatisfactory, due probably to the fact that by the time symptoms appear the toxic condition of the central nervous system is already too advanced to be modified by treatment. In the severe cases it seems of no more value than other plans of treatment, and in the more chronic ones with better prognosis other methods seem equally serviceable. To be of use it should be administered as early in the disease as possible, the dose varying with the preparation, and being still unsettled. My only experience with it has been in subacute cases where sedative drugs were employed as well. In tetanus neonatorum isolated cases of recovery have been reported under this treatment, and there are those⁴ who consider it of undoubted value even when symptoms have already developed. The antitoxin should be given subcutaneously or intraspinally and intravenously, and the treatment should certainly be tried whenever possible. There is good reason to believe that the serum is of value when given as a preventive measure to infants who have been exposed.

CHAPTER XVIII

ACUTE POLIOMYELITIS

(Acute Infantile Paralysis; Meningo-encephalo-myelitis)

History.—There have been in the last few years such changes in our knowledge of this disease that we are now forced beyond question to place it among the acute infectious disorders.

We have no account of it until that of Underwood⁵ in 1784, who,

¹ Jour. Amer. Med. Assoc., 1916, LXVI, 931.

² Arch. of Int. Med., 1916, XVII, 677.

³ Deut. med. Wochenschr., 1890, XVI, 1113.

⁴ See Irons, Jour. Amer. Med. Assoc., 1915, LXIV, 1552.

⁵ Diseases of Children, 1789, Second edition, II, 53.

however, confessed entire ignorance of its cause and considered it not a common disorder. It was not until 1840 that a satisfactory description was given of it by J. Heine.¹ The disease was called the "essential paralysis of children" by Rilliet and Barthez² in 1843. The association with lesions of the anterior horns of the spinal cord was shown by Prévost³ in 1865 and afterward, as a result of careful investigations, by Charcot and Joffroy⁴ in 1870. The first study of its epidemic relations was made in Norway and Sweden especially by Medin.⁵ As a result some writers follow Wickman⁶ in naming it the "Heine-Medin disease." Upon the basis of the newer recognition of its pathology it is properly denominated meningo-encephalo-myelitis, but as this name is cumbersome the older title "poliomyelitis" (*πολιος*, grey) may be retained as representing the more common seat of the lesion; or that of "acute infantile paralysis," since the great majority of cases of acute paralysis in early life are to be classified here.

Formerly a malady of no great frequency as compared with many others, in more recent years its prevalence has increased enormously and the disease has taken its place among the common and serious disorders of early life.

Etiology. Predisposing Causes.—Of the predisposing causes, age is of great importance. The great majority of cases occur between the ages of 1 and 5 years and especially in the 2d year of life. The following table by Frost,⁷ illustrates this well:

TABLE 71.—INCIDENCE OF POLIOMYELITIS—UNITED STATES

	New York Commission 729 cases percentage of total	Massachusetts Commission 615 cases percentage of total	Minnesota Commission 324 cases percentage of total
Under 1 year.....	8.50	7.20	6.50
1 to 5 years.....	82.00	64.50	48.60
6 to 10 years.....	6.40	15.90	23.70
11 to 15 years.....	1.90	5.00	7.70
16 to 20 years.....	0.68	2.4	6.50
Over 20 years.....	0.40	5.0	7.0

European statistics give similar figures, although the percentage among adults is somewhat increased. This is well shown in the following table condensed from one given by Wickman.⁸

TABLE 72.—INCIDENCE OF POLIOMYELITIS—SWEDEN

	1025 Cases percentage of total
0 to 3 years.....	17.85
3 to 6 years.....	20.88
6 to 9 years.....	17.46
9 to 12 years.....	12.00
12 to 15 years.....	10.35
Over 15 years.....	21.46

¹ Beobachtungen ü. Lähmungszustände der unteren Extremitäten. Stuttgart, 1840.

² Maladies des enfants, 1843, II, 335.

³ Comptes rend. de la soc. de biol., 1865, II, 215.

⁴ Arch. de physiol. norm. et pathol., 1870, III, 132.

⁵ Nord. Med. Arkiv., 1896, VI, No. 1.

⁶ Beiträge zur Kenntniss der Heine-Medinschen Krankheit, 1907.

⁷ Public Health Bulletin, 1911, No. 44.

⁸ Die acute Poliomyelitis, Berlin, 1911, 11.

Although less common in the 1st year, it may exceptionally occur even in very early life. Sinkler¹ observed 1 case developing at 6 weeks, and 2 at 3 months; and still younger cases are recorded. A comparative study of the reports of a number of observers show a slight preponderance of the disease among males (Wickman).²

Season also is an important predisposing factor. Everywhere the majority of cases occur in the summer time, the height of an epidemic in the northern hemisphere being July, August and September, the number diminishing rapidly after this; in the southern hemisphere February, March and April. *Geographical position* has no real influence, for although some parts of the world have suffered from it much more than others, the disease is now widely spread. *Residence and social position* are without much influence, poliomyelitis occurring in both cities and rural districts, although somewhat more frequent in the latter in proportion to the population. Other circumstances were formerly supposed to predispose, such as the occurrence of other infectious disorders, exposure to cold, over exercise, and the like; but their influence seems problematical. The *individual susceptibility* is not great. The majority of those exposed, as far as this can be determined, do not contract the disorder. Herrman³ estimates that only about 2 per cent. of children exposed acquire the disease. It is unusual that more than one child in a family is affected. To this there are exceptions, however, especially during epidemics. Notable here is the record of the Swedish epidemic, in which repeatedly more than one child in a house was attacked by the disease. Of 1031 cases reported by Wickman⁴ 627 houses had 1 case each; 95 houses 2 cases each; 39 houses 3 cases; 14 houses 4 cases; 7 houses 5 cases; 1 house 6 cases.

The *epidemic influence* has already been alluded to. Early in its history poliomyelitis occurred chiefly sporadically, and no thought of its infectious or epidemic nature was entertained. The first local epidemic recorded appears to have been one of 8 or 10 cases in a rural region of Louisiana reported by Colmer.⁵ The first epidemic in Norway is said by Harbitz⁶ to have consisted of 14 cases observed by Bull in 1868. After this, at intervals, small epidemics were recorded in different parts of the world, each confined to a small area and limited in number; much the largest being in the Otter Creek Valley of Vermont in 1894, with 123 cases (Calverly).^{7,8} There were frequent local outbreaks in Scandinavia. In 1887 an epidemic of 43 cases occurred in Stockholm and was fully described by Medin.⁹ In 1905 the disease became rather widespread over Norway and Sweden—719 cases being recorded by Harbitz and Scheel¹⁰ in Norway and 1025 cases by Wickman¹¹ in Sweden. Up to 1907 Holt and Bartlett¹² were able to collect a total of 35 reported epidemics, chiefly small, numbering in all about 2000 cases; the majority of these in Scandi-

¹ Keating's Cyclopaedia Diseases of Children, 1890, IV, 685.

² Die acute Poliomyelitis, 1911, 12.

³ Jour. Amer. Med. Assoc., 1917, LXIX, 163.

⁴ Beiträge zur Kenntniss der Heine-Medinschen Krankheit, 1907, 267.

⁵ Amer. Journ. Med. Sciences, 1843, V, 248.

⁶ Journ. Amer. Med. Assoc., 1912, LIX, 782.

⁷ Yale Med. Journ., 1894, I, 1.

⁸ A later report (Journ. Amer. Med. Assoc., 1896, XXVI, 1) gives the number of cases as 132.

⁹ Nord. Med. Ark., 1896, VI, No. 1.

¹⁰ Die acute Poliomyelitis u. verwandte Krankheiten, 1907.

¹¹ Beiträge z. Kenntniss der Heine-Medinschen Krankheit., 1907.

¹² Amer. Journ. Med. Sci., 1898, CXXV, 647.

navia. After this year the number of epidemics and the total number of cases increased rapidly. In 1907 about 2500 cases occurred in New York City and vicinity.¹ In 1909 the disease spread rapidly over many parts of the United States, approximately over 9000 cases in 43 different States being reported in 1910 (Lovett and Richardson).² Meanwhile other epidemics of less size were observed in Australia, Scandinavia, England, Denmark, Cuba, Canada, Germany, and elsewhere, but nothing to be compared with the frequency of the disease in the United States, with the exception of Scandinavia. Here, according to Wernstedt,³ a second great epidemic occurred in Sweden, from the beginning of 1911 to September 15, 1912 more than 6000 cases being reported; while in Norway in 1911, Johannessen⁴ states there were not less than 1407 cases. In the summer of 1916 another very large epidemic occurred in the United States, chiefly in the northeastern portion, the principal locality being New York City and vicinity, in the city itself nearly 9000 cases being reported. In the same summer about 1000 cases occurred in Philadelphia (Le Boutillier).⁵ In all there were about 24,000 cases in the United States in this epidemic (Emerson).⁶ In 1917 there were at least 35,000 cases in the United States (Lavinder).⁷

Exciting Cause.—That the disease is an infectious one seems to have been first suggested by Strümpell,⁸ and all recent clinical experience together with animal experimentation has proven the truth of this. The nature of the infectious element, however, has not been at all understood until within a few years. Of the many earlier bacteriological investigations, one of the most important was made by Geirsvold⁹ who described a diplococcus found in the spinal fluid and the organs. Yet later investigations show that, whatever its significance, this is not the causative factor. In 1909 Landsteiner and Popper¹⁰ injected into the peritoneal cavity of two monkeys the emulsified spinal cord of a fatal case of poliomyelitis, and succeeded in producing the disease in these animals. In the same year Flexner and Lewis¹¹ also produced the disease in monkeys, and were able in addition to transmit it indefinitely through a series of these animals. Later studies by Flexner and Lewis¹² showed that the virus was filterable, passing through a porcelain filter and consequently containing a germ which necessarily is exceedingly minute. That it is, however, a true germ was proven by the fact that the virus is destroyed by heat and by weak disinfectant solutions, such as peroxide of hydrogen, permanganate of potash, and menthol; and that it required a certain incubative time in animal experimentation for the development of symptoms. It withstands the action of glycerine and of freezing; and Römer and Joseph¹³ found that it resisted the effect of drying for as long as 28 days. The virus has not been successfully transmitted to other animals

¹ Report on the New York Epidemic; by the Collective Investigation Committee, 1910, 27.

² Infantile Paralysis in Massachusetts during 1910, 55; 57.

³ *Jahrb. f. Kinderh.*, 1912, LXXVI, 605.

⁴ *Jahrb. f. Kinderh.*, 1912, LXXVI, 603.

⁵ *Amer. Journ. Med. Sci.*, 1917, CLIII, 188.

⁶ *Bull. Johns Hopk. Hosp.*, 1917, XXVIII, 131.

⁷ *Bost. Med. and Surg. Journ.*, 1918, CLXXVIII, 747.

⁸ *Jahrb. f. Kinderh.*, 1885, XXII, 173.

⁹ *Norsk. Magazin for Laegevidskaben*, 1905, LXVI, 1280.

¹⁰ *Zeitsch. f. Immunitätsforschung, Orig.*, 1909, 377.

¹¹ *Journ. Amer. Med. Assoc.*, 1909, LIII, 1639.

¹² *Journ. Amer. Med. Assoc.*, 1909, LIII, 2095.

¹³ *Münch. med. Wochenschr.*, 1910, LVII, 568, 945.

than monkeys, with the possible exception of rabbits (Krause and Meinicke).¹ Most investigators deny the transmissibility to the latter animals. The virus is contained in the brain and spinal cord, salivary and lymphatic glands, tonsils, nasopharyngeal and oral mucous membrane, intestines, and, early in the attack and to a very limited extent, in the blood and cerebrospinal fluid. It can be transmitted experimentally by inoculation into various regions of the body, including the brain, spinal cord, peritoneum and subcutaneous tissue, and can be given by introducing it into the stomach and intestines or by rubbing it upon the nasal mucous membrane. The monkeys never acquire it from each other by mere association. It has been found to persist on the nasal mucous membrane of the animal for as long as 5½ months after the attack is over. Osgood and Lucas² and Pettersson, Kling and Wernstedt³ have found it in the washings from the nose of parents and other attendants upon the patient even 7 months after the occurrence of the attack. The question of the actual nature of the virus is brought nearer solution by the discovery in 1913 by Flexner and Noguchi⁴ in cultures and in the tissues of the brain and spinal cord of very minute globoid bodies in which the power of infection appears to reside, and which would seem to be the actual germ of the disease. It is believed by Rosenau, Towne and Wheeler,⁵ Nuzum and Herzog⁶ and others that the globoid bodies are but a small form of a streptococcus which they have found in the central nervous system of monkeys experimentally infected with the disease. Whether or not the streptococcus is only a secondary infection is not yet definitely determined.

Method of Transmission. Infectivity.—The method of transmission in human beings, and the degree of infectiousness are not clearly understood. It is probable that the virus enters the system by way of the nose—or perhaps the intestine—being acquired from the nasal mucous membrane of an affected individual. Flexner's and Amoss'⁷ experiments support the view that it reaches the nervous system by way of the lymph-channels and without involvement of the blood, and that the portal of entry appears to be the mucous membrane of the upper respiratory tract.

The conditions obtaining are very like those seen in cerebrospinal fever. In both diseases widespread epidemics may occur and the infectivity appear to be much increased; or the disease may develop sporadically and seem to be but little infectious. Both are transmitted in some way from the sick to the well, and probably either directly or indirectly, yet usually not more than one case occurs in a family, and cases of either disease treated in hospitals do not transfer the infection to others in the ward.

That poliomyelitis may be transmitted by unaffected persons acting as carriers has been shown in many instances, notably in the local epidemics reported by Wickman⁸ in Trästäna in Sweden, and by Shidler⁹ in York, Nebraska, respectively. It is uncertain how frequently trans-

¹ Deutsch. med. Wochenschr., 1909, XXXV, 1825.

² Journ. Amer. Med. Assoc., 1911, LVI, 495.

³ 15th Intern. Cong. Hyg. and Demog., 1912, I, 597.

⁴ Journ. Amer. Med. Assoc., 1913, LX, 362.

⁵ Journ. Amer. Med. Assoc., 1916, LXXVII, 1202.

⁶ Journ. Amer. Med. Assoc., 1916, LXXVII, 1205; 1437.

⁷ Journ. Exper. Med., 1914, XX, 249.

⁸ Beiträge z. Kenntniss der Heine-Medicinischen Krankheit., 1907, 150.

⁹ Journ. Amer. Med. Assoc., 1910, LIV, 277.

mission occurs in this way. Very rarely cases have developed after occupying a house which had recently sheltered a patient; yet generally it is impossible to trace the origin of any case to contact with some previous one. This raises the question of the possibility of the spread of the disease by such other agencies as dust (Neustaedter and Thro),¹ bed bugs, (Howard and Clark),² houseflies (Flexner and Clark),³ domestic animals and the like. That some or all of these are factors of importance is possible. Rosenau⁴ is confirmed by Anderson and Frost⁵ in his belief that the infection is communicated chiefly by the bite of the stable-fly. That it is given by the bite of this or any other insect has, however, been disputed by most investigators.

At what period of the disease the infectiousness is greatest is still unknown. That it may persist for a considerable time after convalescence has already been pointed out (5½ months; Osgood and Lucas).

Pathological Anatomy.—This has been studied with especial care in human beings by Wickman⁶ and by Harbitz and Scheel;⁷ and in monkeys by Langsteiner and Popper,⁸ Flexner⁹ and others. In brief, there is a disseminated lymphocytic infiltration of the pia and of all parts of the central nervous system, but chiefly in most instances of the grey matter of the cord, especially in the anterior horns. The changes are most marked in the lumbar and cervical enlargements. The earliest alterations in the spinal cord consist of congestion of the blood-vessels of the pia and nervous tissue, with small-celled infiltration and edema. Hemorrhages, small or more diffuse, sometimes occur into the grey matter. These changes are rapidly followed by degeneration and disappearance of the ganglion cells of the grey matter, the result of the pressure by the inflammatory exudate and edema with the consequent interference with the circulation. The degree and extent of the affection of the ganglion cells varies greatly, dependent upon the degree of involvement of the blood-vessels and of the general small-celled infiltration. In some cases but few cells are involved; in others none at all can be found remaining in certain regions.

The white matter of the cord shows similar congestion, edema, and infiltration of the perivascular spaces, although to a much less extent, with small foci of cellular infiltration in the white matter itself. Changes like those in the white matter are seen in the spinal ganglia and sometimes in the anterior nerve-roots. In the medulla and pons there are lesions similar to those in the cord, but usually with less degeneration of the ganglion cells. The cerebellum and cerebral hemispheres are not so often involved, but the changes resemble those described, consisting of congestion and hemorrhages, with small-celled infiltration of the perivascular spaces and of areas of the brain-tissue itself. As already stated, the anterior horns of the lumbar and cervical enlargements of the cord are the favorite seat of the lesions found; but the process is, in fact, a diffuse one, involving perhaps the entire cord, the meninges of the brain and cord, and many parts of the brain itself as well. Further,

¹ Journ. Amer. Med. Assoc., 1912, LIX, 785.

² Journ. Exper. Med., 1912, XVI, 850.

³ Journ. Amer. Med. Assoc., 1911, LVI, 1717.

⁴ Journ. Amer. Med. Assoc., 1912, LIX, 1314.

⁵ U. S. Public Health Rep., 1912, XXVII, pt. 2, 1733.

⁶ Studien ü Poliomylitis acuta. Arbeiten aus dem path. Inst. d. Universit. Helsingfors, 1905, I, 109.

⁷ Path.-anatom. Untersuch. ü. akute Poliomylitis ü. verwandt. Krankh., 1907.

⁸ Zeitsch. f. Immunitätsforsch., Orig., 1909, II, 377.

⁹ Jour Amer. Med. Assoc., 1910, LV, 1105.

exceptions to the usual distribution are not infrequent, and the medulla, cerebellum, or cerebrum may suffer to a greater extent than the spinal cord.

Other organs show alterations in acute cases, these consisting of degenerative changes, such as are seen in any infectious disorder, and situated especially in the liver, lungs, kidneys and myocardium. Hyperplasia of the lymphoid tissue of the intestine, spleen and mesenteric glands is common. The spleen, lymphatic glands and thymus may be much enlarged. Swelling of the tonsils, with purulent secretion, is frequent.

In cases of long-standing, autopsy shows the affected portion of the spinal cord shrunken and the nervous tissue replaced by sclerotic tissue and neuroglia. The anterior nerve-roots and the muscles exhibit degenerative changes and atrophy, fatty and connective tissue replacing the muscular fibres. The degree of these secondary nervous and muscular lesions corresponds to that of the primary involvement of the spinal cord. Consequently in the milder cases entirely healthy nervous and muscular fibres may be found associated with those more or less atrophied.

Symptoms.—A number of distinct types have been described in addition to the ordinary spinal form which has long been well recognized. Of the various classifications proposed, one frequently adopted is that of Wickman¹ which divides the types into: (1) Ordinary spinal form; (2) progressive form; (3) bulbar form; (4) acute encephalitic form; (5) ataxic form; (6) meningitic form; (7) polyneuritic form; (8) abortive form. These types shade into each other, or may be combined to such an extent that a sharp differentiation may be impossible.

1. SPINAL FORM.—With the exception of the abortive form this is much the most frequent variety, 88.65 per cent. of Zappert's² 555 cases belonging to this category. Many of the statements which follow upon such topics as incubation, method of invasion, mortality, and many of the symptoms of the attack, and its duration and prognosis apply equally well to all forms of the disease.

Incubation.—This would appear to average a week or less, with a range of from 5 to 10 days or exceptionally shorter or longer. In experimental work upon monkeys the incubation may vary through a still greater range. There are no symptoms during this period.

Stage of Invasion.—The onset is usually sudden, with fever, headache, prostration, and occasionally convulsions; as may occur in any of the infectious diseases. Vomiting is a frequent early, or even initial symptom, but is seldom often repeated. Constipation is the rule, but in some cases there is very severe diarrhea. The combination of the latter with vomiting is suggestive of some gastrointestinal affection. In many cases respiratory symptoms predominate, with coryza, conjunctivitis, or bronchitis, and including sore throat. Respiratory symptoms were most prominent in over half of Müller's³ cases, but in the 400 cases analyzed by Wilson⁴ they were not of frequent occurrence. Decided general hyperesthesia, sweating, and great nervous irritability are common and suggestive and headache is generally present. The mind may be clear or there may be somnolence or occasionally coma. An early persistent drowsiness was present in 72 per cent. of Wilson's series. It is accompanied often by a remarkable degree of irritability when the

¹ Beiträge z. Kenntniss der Heine-Medinschen Krankheit, 1907, 10.

² Studien ü. d. Heine-Medinsche Krankheit, 1911, 31.

³ E. Müller, Die Spinale Kinderlähmung, Berlin, 1910.

⁴ Arch. of Pediat., 1916, XXXIII, 850.

child is disturbed. There is very commonly tenderness and stiffness of the neck and back, and pain on moving the body. The tendon-reflexes may in some cases be temporarily increased, but are soon lost. The temperature varies, ranging from 102° to 103° F. (38.9° to 39.4° C.) or occasionally higher; sometimes continuous; sometimes remittent; sometimes falling when the paralysis appears; and continuing from 1 to 10 days (Fig. 162) with an average of 4 days (Wilson)¹ and falling by lysis or by crisis. In the mildest cases fever may be slight and of short duration or occasionally absent. Yet, on the other hand, severe, fatal cases may exhibit

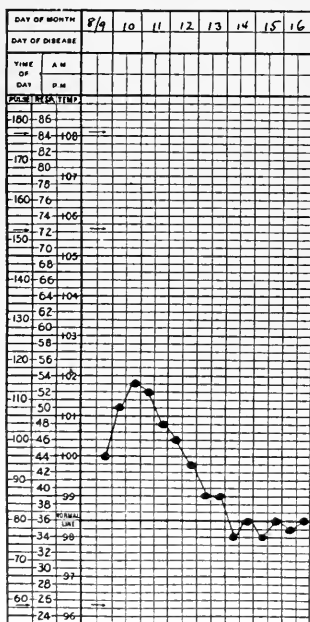


FIG. 162.

FIG. 162.—POLIOMYELITIS, ORDINARY SPINAL FORM.

S. D., girl, aged 6 years. Aug. 9, taken ill with loss of appetite, restlessness, constipation, tenderness and rigidity in neck, sweating of head, twitching and fever; Aug. 10, fever continued, weakness in both legs; Aug. 11, both legs absolutely paralyzed. Cerebrospinal fluid obtained under no pressure showed 36 cells to the c. mm. (Courtesy of Dr. M. Ostheimer.)

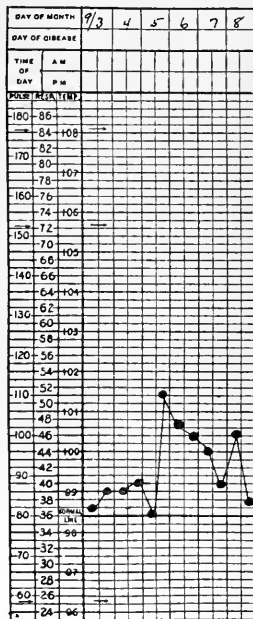


FIG. 163.

FIG. 163.—POLIOMYELITIS WITH LITTLE FEVER. DEATH.

Boy, aged 4 months. Extensive paralysis. Death on the 4th day. (Wickmann, *Beiträge z. Kenntniss d. Heine-Medinsch. Krankh.*, 1907; 15.)

but little rise of temperature (Figs. 163 and 166) and those of much less severity may run a high febrile course (Fig. 164). The temperature in this disease is, in fact, entirely uncharacteristic. (See temperature charts.) Yet more or less elevation of temperature is nearly always present. The pulse is accelerated, often out of proportion to the fever. The urinary functions are generally undisturbed, but retention is not infrequent, while incontinence is uncommon. Zingher² has pointed out the fact that there is an unusual susceptibility to the Shick test in subjects with

¹ *Loc. cit.*² *Amer. Jour. Dis. Child.*, 1917, XIII, 247.

poliomyelitis. Examination of the *blood* gives inconstant results. Occasionally there is decided leucopenia, with slight increase of the lymphocytes; but oftener this condition is absent and there is a moderate or even decided leucocytosis. Examination of the *spinal fluid* of the monkey 24 hours after inoculation, as demonstrated by Flexner and Lewis,¹ and of human cases very early in the prodromal stage shows a moderate increase in the number of cells, especially those of the polymorphonuclear type, which equal 80 per cent. or 90 per cent. of the total number. Promptly, however, before paralysis develops, the lymphocytes become predominant and soon number 90 per cent. or more of the cellular element. The total number of cells ranges from 15 or 20 up to 100, or exceptionally much more in the cubic millimeter; in contradistinction to normal serum which contains only 5 or 10 cells. The fluid is clear or opalescent, forms a fibrin clot in some instances (Kolmer, *et al.*)² and exhibits a moderate reaction for globulin. There is a prompt reduction of Fehling's solution. The cellular increase rapidly diminishes after paralysis has become evident, and by the end of the 2d week the numbers are normal. The globulin diminishes rather more slowly.

The *duration* of the period of invasion varies. The average may be placed at 3 or 4 days, but there are many instances in which the prodromal symptoms are wanting, or so slight that they have been overlooked, and the paralysis comes on suddenly while the child is walking or sitting. In other cases the constitutional symptoms last only during the night and the child is found to be paralyzed in the morning. In still others symptoms may continue for a week before the paralysis appears. It has been pointed out by Kling and Levaditi³ that there is not infrequently a free interval of from 1 to 3 days between certain transitory uncharacteristic symptoms and the onset of what are generally regarded as the symptoms of invasion.

Stage of Acute Paralysis.—At the close of the stage of invasion paralysis shows itself; generally at first as a decided weakness of some of the muscles, but increasing in degree and extent during 3 or 4 days. Exceptionally the development is much slower than this. The regions affected

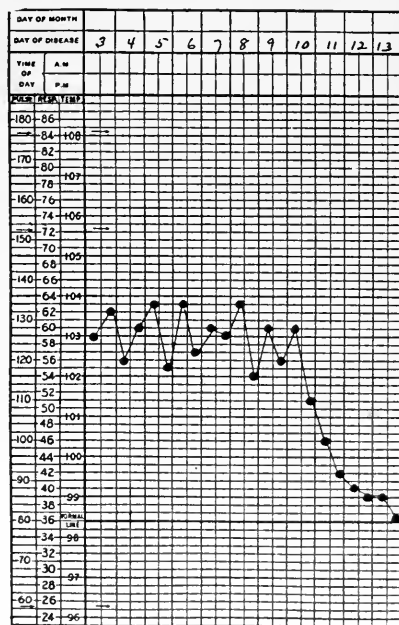


FIG. 164.—POLIOMYELITIS, SPINAL TYPE. SEVERE INITIAL SYMPTOMS.

E. D., male, aged 2 years. Taken ill with loss of appetite, fever, great restlessness, profuse sweating, hyperesthesia and pain on passive movement, slight angina. Paralysis appeared on the 3d day. Recovered with residual paralysis. (Müller, *Die spinale Kinderlähmung*, 1910, 45; 74.)

¹ Journ. of Exper. Med., 1910, March, 227.

² Amer. Jour. Med. Sci., 1917, CLIV, 720.

³ Pub. de l'institut Pasteur de Paris, 1913. Ref., Haynes, Arch. of Pediat., 1917, XXXIV, 401.

vary greatly. Much most frequently the paralysis is situated in one or both legs, the parts below the knee being oftener or more severely involved than those above. Not all the muscles of the limbs are affected, the peroneal muscles and the tibialis anticus and posticus being especially prone to be attacked. So, too, not all the fibres of any muscle participate in the process. Paralysis of one upper and one lower extremity is not uncommon, as is also a general paralysis of all four limbs, with some involvement of the neck and trunk. Less often one arm is attacked, the lower extremities being spared; and still less often both arms only. When the extremities of both sides of the body are involved, one side is almost always more severely affected than the other.

As already stated, there may be a combination of some of the different forms of the disease. Thus it occasionally happens that paralysis of some of the extremities occurs in combination with a facial palsy. Some idea of the frequency of paralysis in different parts of the body may be obtained from the following statistics condensed from figures given by Lovett and Richardson,¹ based on 1158 cases:

TABLE 73.—REGIONS PARALYZED IN POLIOMYELITIS—AMERICA

	Per cent.
One leg only.....	27.97
Both legs.....	23.48
Both legs and arms.....	11.13
One leg and one arm, same side.....	9.49
One arm only.....	7.25
Both arms only.....	1.98
Face.....	6.38
Abdomen.....	5.78
Neck.....	0.94
Respiration.....	2.67
Deglutition.....	0.60

European statistics may be compared by consulting the analysis given by Wickman,² but reduced to percentages, of his 868 cases with paralysis.

TABLE 74.—REGIONS PARALYZED IN POLIOMYELITIS—SWEDEN

	Per cent.
One or both legs.....	40.67
One or both arms.....	8.64
Combined paralysis of arms and legs.....	17.51
Legs and trunk.....	9.79
Arms and trunk.....	1.15
Trunk alone.....	1.04
"Whole body".....	2.65
Ascending paralysis.....	3.69
Descending paralysis.....	1.50
Combined spinal and cerebral nerves.....	3.92
Cerebral nerves alone.....	2.65
Localization not defined.....	6.91

The *constitutional symptoms* of the stage of invasion persist during the first 3 or 4 days of the acute paralytic stage, but last very seldom more than 6 or 7 days in all from the onset, and often a decidedly shorter time. Pain, a frequent symptom, may, however, continue several weeks. There is no anesthesia. Generally after cessation of acute

¹ Infantile Paralysis in Massachusetts during 1910, 92.

² Die acute Poliomyelitis, 1911, 44.

constitutional symptoms there is no addition to the paralysis. To this there are sometimes exceptions seen, and paralysis continues to increase for a few days longer. The condition of the spinal fluid in this stage has already been considered (p. 525).

Stationary Stage.—This follows the cessation of acute symptoms and lasts from 1 to 6 weeks. No change takes place in the paralytic condition except that atrophy rapidly develops; and no other symptoms of any sort are present, except in some cases a decided persistence of a degree of pain and of hyperesthesia. During the stationary period the paralysis seems often very extensive and complete. Paralysis of the muscles of the trunk is not infrequent; even those of the neck and abdomen and the muscles of respiration being more or less involved. In such cases of extensive paralysis the child lies helpless in bed, and if lifted to a sitting position is unable to support the body or to hold the head erect. It is impossible at this period to predict how complete the paralysis will remain.

Stage of Retrogression.—At the end of the stationary period improvement begins. The greater part of this will occur in the first 6 months, less in the next half year, but some degree takes place even up to the end of 2 years. Meanwhile atrophy becomes still more distinct in the muscular tissue which is not undergoing improvement, and by 2 months is very decided. The affected limb is then usually much smaller than normal in circumference, and peculiarly soft and flabby. There is no tactile anesthesia or analgesia. The paralysis is flaccid in nature (Fig. 165), there being diminished muscle-tonus and diminution or absence of tendon reflexes.

Cases which at first seemed severely paralyzed improve usually greatly during this period of retrogression. Probably in these cases the muscular paralysis is dependent chiefly upon the pressure of the edema and of the cellular infiltration. Those muscle-fibres connected with ganglion cells which have been destroyed can never regain their power. It is not possible early to determine how much of such actual destruction has taken place.

Chronic Atrophic Stage.—In the final condition the growth of the affected limb is much interfered with, the limb being both smaller in circumference and shorter than normal, the muscles greatly wasted and the ligaments much relaxed, allowing deformities to result from yielding of the articulations. The skin is cold, blue, and marbled, and the patient suffers from a sensation of coldness in the paralyzed parts. Contractions commence even during the stage of retrogression, and in the chronic stage are well developed, the stronger muscles overcoming

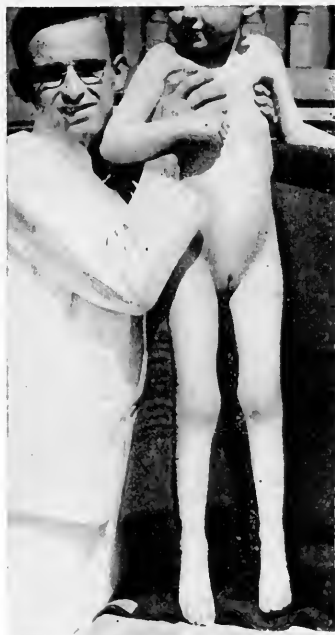


FIG. 165.—PARALYSIS IN POLIO-MYELITIS.

Recent case in girl of 6 years. Complete paralysis in the lower extremities, with foot-drop. From a patient in the Children's Hospital, Philadelphia.

the weaker paralyzed ones. Talipes and lateral spinal curvature are among the commoner of the deformities. The parts of the body oftenest showing residual paralysis are one or both lower extremities, one limb being always worse than the other. Next most frequent is one upper, or one upper and one lower extremity. Thus the muscles oftenest and most severely invaded in the primary paralysis are those which are left paralyzed finally.

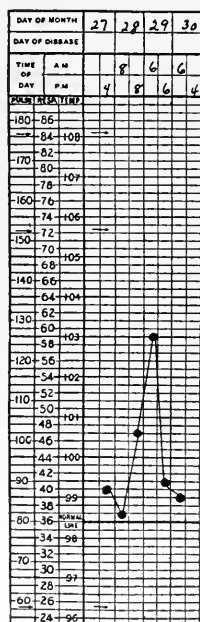


FIG. 166.

FIG. 166.—POLIO-MYELITIS. PROGRESSIVE FORM.

E J., male, aged 10 years. Aug. 27, fever, malaise, headache; Aug. 28, slight restlessness; Aug. 29, nervous, head-sweating, neck rigid, fever. Weakness of left leg in morning, entirely paralyzed by noon. Right leg weak at 4 p. m., totally paralyzed at 8 p. m. Upper extremities and shoulder muscles paralyzed by evening; Aug. 30, paralysis of muscles of deglutition and respiration. Cerebrospinal fluid under increased pressure, 94 cells to the c.mm. Child conscious until 2 p. m., died at 5 p. m. *Courtesy of Dr. M. Ostheimer.*

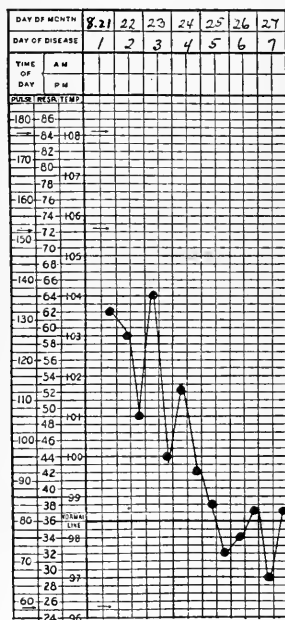


FIG. 167.

FIG. 167.—POLIO-MYELITIS, BULBAR FORM.

Charles J., aged 4 years. Aug. 21. Tonsillitis on the 17th. Fever disappeared but returned today, with nervousness, dullness, vomiting, loss of appetite, dry tongue, constipation; Aug. 22, nearly comatose, muscular twitching, tympanites, high fever, increased knee-jerks, pain in arms and legs, slight paralysis of face. *Later.*—Little change for 5 days, except increasing paralysis of the right side of the face, and apparently some difficulty in swallowing. Then gradual improvement in all symptoms, with loss of knee-jerks. Very decided paralysis of the face remained, with slight weakness in the right leg.

Electrical Reactions.—The importance of this matter makes it advisable to consider together the conditions observed in the different stages. By the beginning of the 2d week of the attack both faradic and galvanic contractility are lost in the severely paralyzed muscles. Very soon, oftenest in the course of the 2d week, there is a reappearance and then an increase in the galvanic response, with the presence of the reaction of degeneration. The galvanic contractility then diminishes, but still with the degenerative reaction which may last for some months.

Finally, perhaps after 2 or 3 years, all electrical contractility of any sort disappears in the permanently paralyzed muscles. In those which recover, faradic contractility slowly reappears and the galvanic returns to a normal condition. In muscles but little affected faradic contractility may never be more than merely diminished.

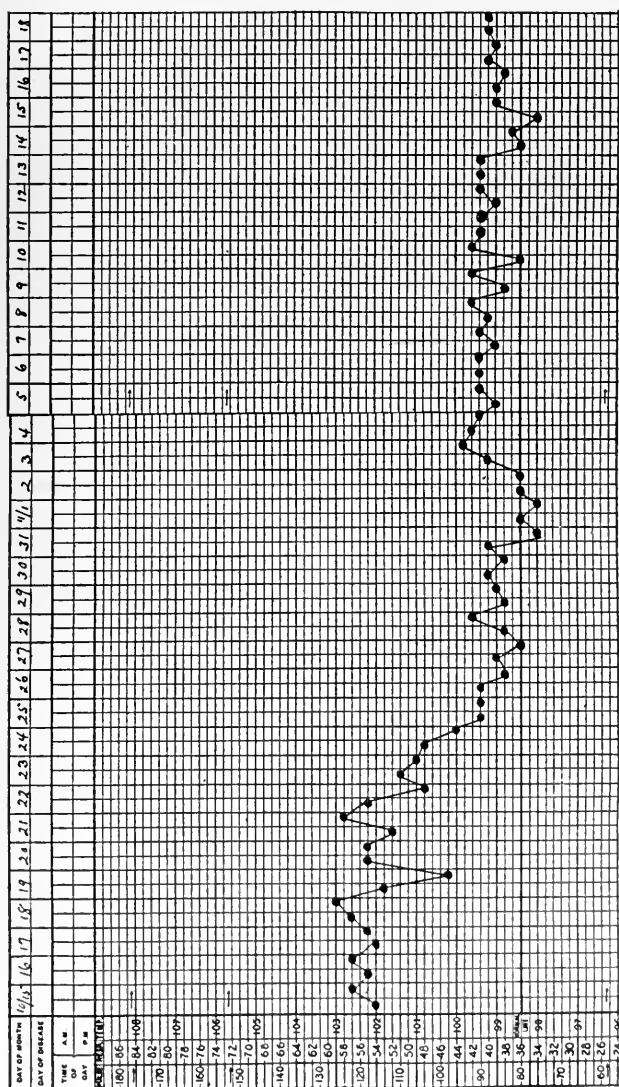


FIG. 168.—POLIOMYELITIS, BULBO-SPINAL FORM. SUPPOSED TO BE MENINGITIS.

Betty B., aged 3½ years. Taken ill Oct. 15 with fever. Vomited on 2d day. Semicomatose for a week. Slight stiffness of neck, grinding of teeth, tache, occasional sighing respiration. Lumbar puncture done on three occasions gave slightly cloudy fluid in increased amount, increase of polymorphonuclear cells (90 per cent.), no germs. Anti-meningococcal serum injected on ground that the case might be cerebrospinal fever. Not until convalescence began was there found extensive paralysis of the right upper extremity and to a less extent of the left, and transient paralysis of left external rectus and left side of face.

2. PROGRESSIVE FORM (*Landry's Paralysis Type*).—This form, not a common one, is characterized by the rapid extension of the lesions until the medulla is involved. It was seen in 45 (4.39 per cent.) of Wickman's¹ 1025 cases. The paralysis generally begins in the lower extremities and ascends to the arms, involving the muscles of the trunk

¹ Die acute Poliomyelitis, 1911, 53.

including those of respiration, and sometimes the diaphragm and even the muscles of deglutition. The course is rapid, death following in a few days (Fig. 166). Sometimes the arms are attacked first, and the extension is then of the descending type. It is probable that the majority of the cases which have been described as Landry's paralysis should be grouped here. (See Landry's Paralysis, Vol. II, p. 382.)

3. THE BULBAR OR PONTINE FORM.—Formerly this condition was described as a distinct disease under the title of "Polioencephalitis superior or inferior," according to which of the nuclei were involved.



FIG. 169.—COMBINED LESIONS IN POLIOMYELITIS.

Infant of 3 months in the Children's Hospital of Philadelphia. Facial palsy, flaccid paralysis of the legs, spastic paralysis of the arms.

The lesions may be limited to the nuclei of the cranial nerves, or may be combined with spinal lesions (Figs. 167, 168). The facial nucleus is the one oftenest affected, and the abducent and the hypoglossal frequently; but any of the cranial nerve-nuclei may be involved with the production of the corresponding symptoms, among them central paralysis of respiration, paralysis of deglutition, and disturbances of the heart's action. Although the bulbar paralyses usually recover completely, a transitory paralysis of some muscles of the limbs may sometimes be combined with a permanent facial palsy, and the diagnosis may later be difficult if the early history of the attack is not known. The bulbar form is not infrequently seen, 6.45 per cent. of Wickman's¹ 868 cases belonging here.

4. ACUTE ENCEPHALITIC FORM.—The variety bearing this name was described by Strümpell² as "acute encephalitis or polienccephalitis of children," and its close relationship to, or identity with, poliomyelitis suggested. It is now generally recognized as a variety of poliomyelitis. It is one of the most unusual types of the disease. In it the grey matter of the cortex is probably involved. The early symptoms show great variation but suggest those of meningitis. The disease may begin with convulsions, which may be unilateral, somnolence develops, and a paralysis appears with *increase of tendon-reflexes*. Later the paralysis is found to be of a spastic type and to some extent of hemiplegic distribution, showing its cerebral origin. This can be combined with flaccid paralysis located in other regions, and due to coincident involvement of the lower segment of the cerebrospinal system (Fig. 169).

5. THE ATAXIC FORM.—This is likewise an unusual form, seen in only 0.43 per cent. of Lovett and Richardson's³ 1158 cases. In it ataxia is the chief symptom, oftenest of the lower extremities, or being more extensive; and this either occurs alone or is combined with cerebral symptoms, especially paralysis of the cranial nerves, and sometimes with a moderate

¹ Die acute Poliomyelitis, 1911, 44.

² Jahrbuch für Kinderheilkunde, 1885, XXII, 173.

³ Loc. cit

degree of spinal paralysis as well. Cases of acute cerebellar ataxia are probably in most instances to be classed here. (See Cerebellar Ataxia, Vol. II, p. 383.)

6. THE POLYNEURITIC FORM.—The disease in this form simulates multiple neuritis so closely that its existence has been much disputed. It certainly is a rare variety. It is characterized by the severe pain and tenderness in the nerve-trunks and muscles. The pain may be present

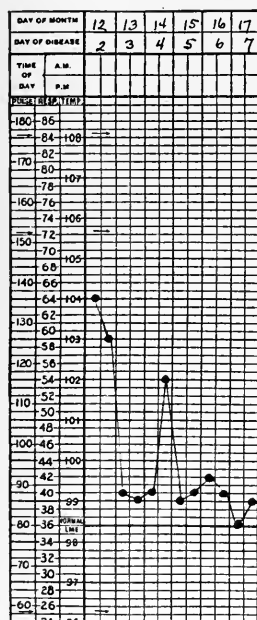


FIG. 170.

FIG. 170.—POLIOMYELITIS, MENINGITIC TYPE.

Boy, aged 8 years. June 11, attacks of headache and convulsions; June 12, somnolence, retraction of head, opisthotonos, pain on movement, rigidity of limbs, at times tremor, patellar reflexes appeared to be increased. Later.—Condition little changed for 4-5 days, then rigidity disappeared, somnolence persisted a few days longer. Recovery. No paralysis at any time. (*Wickmann, Beiträge z. Kennt. d. Heine-Medinsch. Krankh.* 1907, 15; 100.)

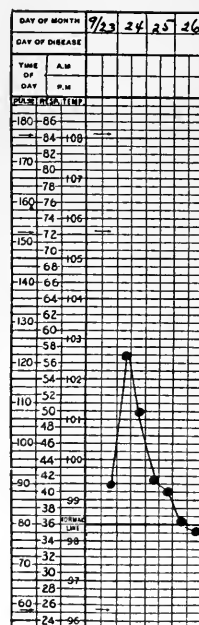


FIG. 171.

FIG. 171.—POLIOMYELITIS, ABORTIVE TYPE.

Walter B., aged 5 years. Illness began Sept. 22, during an epidemic, with fever, stiffness in feet, slight headache, constipation, and loss of appetite; Sept. 23, examination shows child drowsy, feverish, does not answer questions, throat red, slight tache, plantar reflexes slightly exaggerated, other reflexes normal. Spinal fluid under increased pressure, clear, cell-count 30, with 22 polymorphonuclear cells, no organisms; Sept. 24, some jerking of legs and arms; Oct. 2, child convalescing, but difficulty in straightening legs when sitting up, Kernig's sign present both sides, knee-jerks and plantar reflexes slightly exaggerated; Oct. 4, excellent general condition. No suggestion of paralysis.

in the joints as well. The paralysis may sometimes be slight or transitory, or even overlooked, and the limbs held rigidly to guard against pain on movement. In other cases the paralysis is extensive. Disturbances of sensibility are usually absent.

7. THE MENINGITIC FORM (Fig. 170).—In this variety, not uncommon in epidemics, the very marked symptoms indicating implication of the meninges overshadow all other manifestations. There are

vomiting, headache, pain and rigidity in the neck and back even with moderate opisthotonos, convulsions, delirium and coma. Not all these are present in any one case, and combinations of any sort are possible. A diagnosis can be made only if paralysis develops. The cases are often severe and death may occur in a few days.

8. THE ABORTIVE FORM (Fig. 171).—The frequency and even the possibility of such cases have come into prominence only in recent years. Their existence has been proven experimentally by Flexner and Clark,¹ and especially by Anderson and Frost² through the discovery of immunizing bodies in the serum of patients who had suffered from this form of the disease, and by the production of the disease in animals by the inoculation

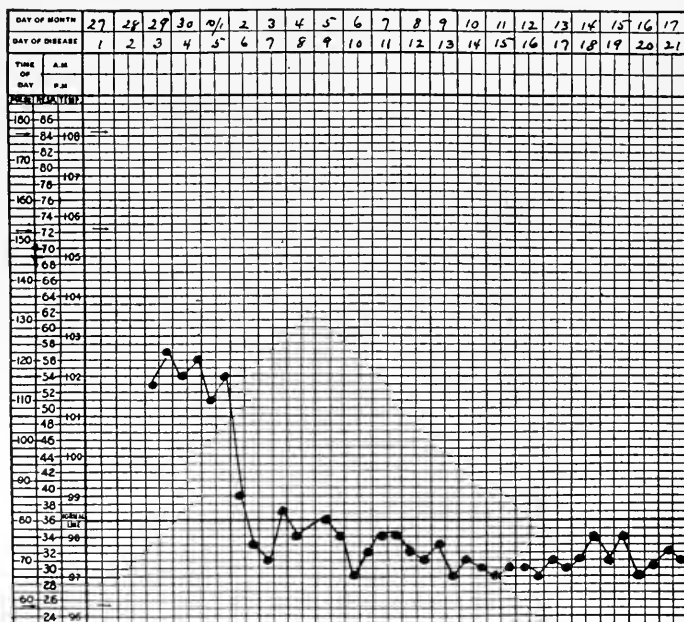


FIG. 172.—POLIOMYELITIS, ABORTIVE (RUDIMENTARY) FORM.

E. B., girl, aged 4 years. Onset with nausea, slight sore throat, headache and fever. Spinal fluid on 5th day showed no increase of pressure, leucocytes 36 to c. mm., with 63 per cent. lymphocytes. Apparently convalescent on 6th day. On 17th day slight weakness in both legs which disappeared by 21st day. *Courtesy of Dr. M. Osheimer.*

with the virus from abortive cases. There is abundant clinical evidence also, as seen in families where some cases are of the typical form, and others have only initial symptoms without any subsequent paralysis. The spinal fluid, too, exhibits the characteristic changes. The attack begins abruptly with the prodromal manifestations already described. There is fever of varying type, usually constipation, prostration, severe headache, and often vomiting. Frequently hyperesthesia is present with stiffness of the neck and pain here and in the back. Sore throat may be present, or rhinitis and conjunctivitis. In some instances there is a muscular weakness or a certain unsteadiness in walking. The character of the symptoms varies decidedly, as in the initial stage of the

¹ *Loc. cit.*, 585.

² *Journ. Amer. Med. Assoc.*, 1911, LVI, 663.

ordinary form. Sometimes gastrointestinal manifestations prevail; sometimes respiratory; sometimes nervous. After continuing 2 or 3 days, there is rapid recovery. To this class of abortive cases might be added what E. Müller¹ has denominated the "*rudimentary form*" (Fig. 172), in which a very slight transitory paralysis is seen with temporary loss of the patellar reflexes. This is in contradistinction to cases without any evidences whatever of paralysis, which he calls the "*larval form*."

Abortive cases are frequent in epidemics and doubtless often overlooked. Müller estimated that one-half of the cases of poliomyelitis are of this type, and of Wickman's² 1025 cases 157; *i.e.* 15.32 per cent., were of the abortive type. In the second great Swedish epidemic the abortive cases in some localities greatly exceeded those with paralysis (Wernstedt),³ and the same is probably true of the 1916 epidemic in the United States.

Relapse.—Occasionally after the development of paralytic symptoms of moderate degree, there is an intermission of 6 or 7 days, after which there is renewed extension of paralysis with reappearance of other symptoms. In some cases this is probably only a later manifestation of the initial lesion; but in others there appears to be a true relapse occurring, it may be weeks after the primary attack. Leegaard⁴ reports a case probably of this nature in which there was an interval of nearly 3 weeks.

Recurrence.—A patient who has once suffered from the disease appears to be immune. Experimental work with monkeys likewise shows that the prompt injection of the blood of other monkeys or of human beings, who have had the disease, will sometimes prevent or delay the development of paralysis after inoculation. The immunizing principle has been found in the blood of individuals several years after the attack of the disease (Flexner and Clark).⁵ There are, however, occasional exceptions to this rule of lasting immunity, and second attacks have been reported, as by Sanz⁶ and by Lucas and Osgood.⁷

Prognosis.—Formerly poliomyelitis was considered a disease not at all dangerous to life, and only to be feared on account of the lifelong crippling which it left. Experience in more recent years shows that under epidemic influences the mortality can be very decided. Thus Krause⁸ reports a mortality of 15.14 per cent. in 436 cases; Zappert⁹ 10.99 per cent. in 555 cases; Lindner and Mally¹⁰ over 16 per cent. in 96 cases; Wickman¹¹ 12.2 per cent. in 1025 cases or 16.7 per cent. if the 157 abortive cases are excluded; Johannessen¹² 13.08 per cent. in 1407 cases; Leegaard¹³ 17.1 per cent. in 3290 cases. The American death-rate has been somewhat less. Lovett and Richardson¹⁴ found a mortality of 7.9 per cent. in 1216 cases in Massachusetts, when in approximately 2000 cases in New York City the mortality was about 5 per cent.¹⁵ A notable exception to the compara-

¹ *Loc. cit.*, 146.

² Beiträge zur Kenntniss der Heine-Medinschen Krankheit, 1907, 288.

³ Wernstedt, Jahrb. f. Kinderh., 1912, LXXVI, 605.

⁴ Nordk. Mag. for Laegevid, 1901, XVI, 377.

⁵ *Loc. cit.*

⁶ El Siglo Med., 1915, LXII, 530. Ref., Brit. Jour. Child. Dis., 1916, XIII, 56.

⁷ Journ. Amer. Med. Assoc., 1913, LX, 1611.

⁸ Deutsche med. Wochenschr., 1909, XXXV, 1822.

⁹ Die Klinik. u. Epidemiologie d. acuten Kinderlähmung.

¹⁰ Zeitschrift für Nervenheilkunde, 1910, XXXVIII, 362.

¹¹ Beiträge zur Kenntniss der Heine-Medinschen Krankheit., 1907, 286.

¹² Jahrb. f. Kinderh., 1912, LXXVI, 603.

¹³ Deut. Zeit. f. Nervenheilk., 1914-15, LIII, 222.

¹⁴ *Loc. cit.*, 78.

¹⁵ Report of New York Collective Investigation Committee, 1910, 21.

tively low mortality-rate was seen in the epidemic in New York State in 1916. In this there were approximately 13,000 cases, with a mortality of 25 per cent (Nicoll).¹ The abortive cases, of course, give an entirely favorable prognosis. Cases of the polyneuritic type may have paralysis persist, or there may be complete recovery. The ataxic symptoms of the form of the disease bearing this name are never more than a temporary matter. The fatal cases are seen especially in the progressive form, which nearly always ends in death, and in the acute encephalitic and the meningitic forms, both of which are serious conditions. The occurrence of complications is a not infrequent cause of death. Among these is to be mentioned especially bronchopneumonia. In the fatal cases death is liable to occur in the first 3 to 5 days, during the continuance of the active constitutional symptoms, and probably certainly in the first 2 weeks of the attack. After this period recovery is almost assured so far as life is concerned. The influence of the age of the patient is very decided. Statistics show that the mortality in children is decidedly less than later. Wickman² found a mortality from 0 to 2 years of 10 per cent. and from 3 to 5 years of 11.6 per cent.; gradually increasing until from 30 to 32 years it was 33.3 per cent.

The likelihood of recovery from the paralytic condition is a matter of prognostic importance. The early paralysis of the trunk and neck seen in the spinal cases will probably disappear. Not infrequently all traces of paralysis everywhere may cease to exist. Lovett and Richardson³ found that in 16.7 per cent. of 150 cases analyzed by them all evidences of the disease had disappeared within 3 months. Wickman⁴ was able to study 530 cases of primary paralysis from 1 to 1½ years after the attack. Of these 56 per cent. still showed paralysis and 44 per cent. none at all. The paralysis in cases of the bulbar type generally disappears completely. Very commonly, however, in the spinal cases some evidence of paralysis remains throughout life. This may vary anywhere from complete disability to a slight impairment of motion, most frequently seen as a trifling limp in walking. The ultimate degree of disability depends often upon the faithfulness with which treatment has been carried out. The muscles of the arms and shoulders seem less liable to undergo recovery than those of the legs. Muscles in which entire paralysis and complete loss of faradic contractility are present after the acute stage is over, in the course of the 2d week, are liable to be more or less permanently paralyzed. If slight return of faradic contractility is found in the course of a few days longer, partial recovery will probably follow. If after 2 or 3 months there is much wasting and loss of power and continued loss of faradic contractility, little improvement can be expected. Muscles which have never exhibited electrical changes will probably recover completely in a few weeks or months. Even in cases seen for the first time in the chronic stage, if there is some degree of faradic contractility remaining, considerable improvement under treatment may be expected.

Diagnosis.—It seems impossible to recognize poliomyelitis with certainty in its prodromal stage. Lumbar puncture made just before paralysis appears shows a clear or slightly opalescent fluid, containing an increase of mononuclear cells, and a moderate globulin reaction. A spinal fluid of this nature may be readily distinguished from that of cere-

¹ Amer. Jour. Dis. Child., 1917, XIV, 69.

² Beiträge zur Kenntniss der Heine-Medinschen Krankheit., 1907, 288.

³ Loc. cit., 70.

⁴ Die acute Poliomyelitis, 1911, 78.

brospinal fever, but not positively from tuberculous meningitis. In the latter the albumin and globulin are generally greater in amount, the sugar diminished or absent, and the cells more numerous. But to this there are so many exceptions that the differences are not trustworthy. Moreover, the symptoms of poliomyelitis are so very indefinite that it would be necessary to perform lumbar puncture on every child with manifestations of a dubious sort. It is only when an epidemic is prevailing that the beginning of poliomyelitis will be thought of and that lumbar puncture will be tried. The most suggestive of the early manifestations are hyperesthesia, sweating, and nervous irritability. The variation of the complex of early symptoms, however, makes a number of different diagnoses possible at this time. Vomiting and constipation suggest a gastrointestinal disorder; severe diarrhea, an intestinal auto-intoxication; severe pain in the head and stiffness of the neck and convulsions, a cerebrospinal fever or other similar form of meningitis; somnolence or coma with rigidity, a tuberculous meningitis; headache, coryza, decided prostration and rapid pulse, gripe. *Cerebrospinal fever* has symptoms very similar to poliomyelitis with decided meningitic symptoms. Lumbar puncture, however, shows a cloudy or even purulent fluid, with numerous polymorphonuclear cells. The blood, too, in the first disease exhibits uniformly a high leucocytosis. Fever is of longer duration in cerebrospinal fever and there is oftener coma. The reflexes are increased and a spastic condition soon develops.

Tuberculous meningitis exhibits a spinal fluid very like that of poliomyelitis and the diagnosis may be impossible for some days. Then, however, the failure of paralysis to appear and the increase of the stuporous condition point to the former malady. The onset of meningitis is usually much more insidious, although to this there are exceptions.

When once paralysis has developed, the diagnosis of poliomyelitis is usually apparent, although it often happens that this condition is not recognized for some time, the helpless state being supposed to be the result of prostration. Even after the stage of retrogression is over, mistakes in diagnosis are still possible. There is scarcely, however, any other form of paralysis which gives the picture of flaccidity, atrophy, and flail-like freedom of movement at the articulations, unless it be *multiple neuritis*. This disease is more frequent than the polyneuritic type of poliomyelitis, the onset is slower, and there are no cerebral symptoms. It is generally much more widespread over the body and there is considerably more pain in the limbs; yet this last feature is not a safe diagnostic sign. A diminution of the touch-sense indicates the presence of neuritis rather than poliomyelitis. The likeness of the two diseases is certainly very close, but the anatomical identity is not yet proven. A *localized neuritis*, oftenest of one arm, as in instances of obstetrical paralysis, sometimes causes error in diagnosis. Only the history of the case can distinguish it. *Infantile scurvy* may readily simulate poliomyelitis through the development of pseudo-paralysis, the result of pain on movement. I have seen the mistake in diagnosis made in a number of instances. The associated symptoms, the history of the case, and the prompt reaction to antiscorbutic treatment soon settle the question. The pseudo-paralysis of severe *rickets* has repeatedly been supposed to be the result of poliomyelitis. A little care in observation will remove all doubt. In rickets there is the history of slow onset and the presence of associated symptoms. There exists a weakness rather than a paralysis, and this is widespread and not more marked on one side than the other.

Electrical reactions also are different. The ordinary *cerebral paralyses* are not easily confounded. They are spastic in character, with exaggerated tendon reflexes. Only in the uncommon acute encephalitic form of poliomyelitis is there a spastic paralysis present. The history of the attack aids in the diagnosis, as would the early employment of lumbar puncture. Even the contractions about the joints in old cases of poliomyelitis are easily recognized not to be spastic in character, but due to actual shortening of the muscles. In instances of the bulbar form of poliomyelitis, facial paralysis may readily be supposed to be a *Bell's palsy*, due to neuritis of the facial nerve. The history of the attack is, however, entirely different and the frequent combination of some paralysis of the limbs makes the matter clear.

Treatment. Prophylaxis.—Until there is greater knowledge of the method by which the disease is contracted, little can be expected in the way of preventive treatment. The fact that the virus is found at times on the nasal mucous membrane, and even may be virulent long after the disease is over, indicates the advisability of thorough frequent disinfection of this region in patients suffering with the disease, in the hope of preventing its dissemination. This brings up, too, the subject of separation and quarantine. In spite of the absence of positive evidence of direct transmission, quarantine should be enforced, especially during an epidemic prevalence of the disease. With a lack of knowledge of the usual period of infectiousness, the proper duration of isolation is a matter of uncertainty. A quarantine of 3 weeks would seem ample, unless inflammation of the nose and throat is present. How much longer would be necessary to ensure absolute safety cannot, as yet, be determined. A child who has been exposed may be considered safe if not developing the disease in 2 weeks. Those who have come into contact with patients should employ disinfection of the nose and throat to prevent the possibility of their acting as carriers; or, still better, wear a mask of gauze or similar substance over the mouth and nose. The discharges from the mouth and nose of the patient should be promptly destroyed. After the room is vacated, it should be thoroughly disinfected. It is also advisable not to take children to localities where the disease is epidemic; or, if already there, to forbid their association with those who have recently had the affection, or with those who are ill with any vague symptoms, as well as to prevent their frequenting public conveyances or public places of amusement where many children are together.

The ability to immunize human beings in times of epidemics would be most desirable, and may be finally accomplished. It has sometimes been done successfully in monkeys by Flexner and Lewis¹ and others, but as yet it is too uncertain for application to human medicine.

Treatment of the Attack.—During the initial stage of the attack there is as yet no treatment of certain avail open to us. In the case of monkeys it has been shown by Flexner and Clark,² that the administration of hexamethylenamine has delayed the development of symptoms, and it is earnestly to be hoped that further investigations along this line may result in a satisfactory treatment. At least no harm can follow the administration of this drug in fairly large, divided doses, of 20 to 40 grains (1.3 to 2.6) daily; but to be of any possible benefit it must be given before paralysis develops.

¹ Journ. Amer. Med. Assoc., 1910, LIV, 1780.

² *Loc. cit.*, 585.

Based upon animal experimentation in other conditions, Meltzer¹ recommended the administration of adrenalin, giving 0.5 c.c. (8 m.) of the 1:1000 solution intraspinally. This had been previously tried by Clark² in monkeys, with considerable success. Netter³ in 1910 used the injection of blood-serum from subjects who had previously suffered from poliomyelitis, and since then has employed it in 32 cases with, he believes, excellent results. In the 1916 epidemic this method of treatment was tried in a considerable number of instances, apparently with good results. Zingher⁴ used it in doses of 10 to 15 c.c. (0.34 to 0.51 fl.oz.) intraspinally every 20 to 24 hours for 2 or 3 days. He reported its employment in 418 cases and claimed a beneficial action when given in the preparalytic stage. Others share the opinion that the results in the preparalytic stage are good, and still others have seen about the same proportion of cases escape paralysis when no serum was employed. The great difficulty in determining the value of any form of treatment in this disease renders further observations necessary before any positive conclusions can be drawn. In the preparalytic stage in experimental poliomyelitis in monkeys the treatment appears to be of undoubted value (Flexner and Amoss).⁵

Complete rest in bed is imperative, but counterirritation with blisters or similar measures merely adds to the discomfort, and there is no evidence that it can be of avail at any stage of the disease. Remedies may be needed to relieve pain, restlessness, or other nervous symptoms; to control fever; and to support strength. It is not yet time for electricity and massage, which can do no good and may do harm. All such treatment should be delayed until the stationary stage is over and all tenderness has disappeared. This may not be for from 3 to 6 weeks or longer after the onset. The value of electricity and massage is to improve by this enforced exercise the tone and the power of the muscles which have been only partially or temporarily injured. Those muscle-fibres presided over by absolutely atrophic ganglion-cells can never show any improvement. The faradic current should be applied to the different muscles which have been paralyzed, continuing the entire treatment for from 10 to 20 minutes, once or twice a day. If there is no response to faradism, galvanism may be tried. Only such strength of current of either sort should be used as will produce moderate contractions without pain. Massage and passive movements can be commenced as soon as the acute stage is over and all hyperesthesia and pain have disappeared. It is very important to combine with the passive exercise the encouraging of the patient to make voluntary active movements, no matter how imperfectly these are made at first. More can be expected from this than from any other plan of treatment, and if persevered with for months or even for years remarkable improvement will often follow in cases which at first appeared most discouraging. Confinement to bed should continue for 2 or 3 months except in the mild cases, when 2 or 3 weeks may be sufficient. As a result of the study of a large number of cases—1836—Lovett⁶ emphasizes the risk attending the permitting of any weight-

¹ Med. Rec., 1916, XC, 171.

² Journ. Amer. Med. Assoc., 1912, LIX, 367.

³ Arch. de méd. des enf., 1916, XIX, 1.

⁴ Arch. of Pediat., 1916, XXXIII, 872.

⁵ Journ. Exper. Med., 1917, XXV, 499.

⁶ Jour. Amer. Med. Assoc., 1917, LXIX, 168.

bearing exercise for a long period after the attack, and shows that there is danger of increasing the degree of residual paralysis already present.

Mechanical Treatment.—It is important to prevent deformity by the employment as early as necessary of mechanical apparatus. Even during the early part of the stage of retrogression one must guard against beginning deformity. Sand-bags at the soles of the feet or the wearing of a shoe with elastic extending from the toe to the hip will aid in preventing the contraction of the tendo achillis, which is so likely to occur. A proper position in bed is necessary, too, in many cases to avoid the production of lateral curvature. While the use of braces is to be deprecated on the ground that it adds to the weight to be moved and removes the necessity for the paralyzed muscle to do its work completely; yet, on the other hand, it is still worse to allow deformities of any part to develop. The need for their employment must be determined for the individual case. Sole dependence upon crutches is harmful, as it permits the muscles to remain in an entirely unused condition. Tenotomies will be required less frequently if measures are early undertaken to prevent contractions forming. In some cases tendon-transplantation may be performed with benefit. This, as a rule, is best deferred for at least 2 years. In other cases artificial ankylosis may give necessary rigidity to an otherwise useless joint. The great principle of treatment in the chronic paralytic stage is never to abandon it, since slow improvement may finally come, and aid may be given in some way in cases at first most unpromising.

CHAPTER XIX

TUBERCULOSIS

Tuberculosis affects many different parts of the body, the local symptoms usually greatly preponderating over the general ones, and producing clinical pictures essentially different from each other. On this account, and for the sake of greater convenience of study, some, at least, of the special forms of tuberculosis will be discussed more in detail in different sections. In the present chapter tuberculosis as a whole will be considered, especially as it affects children, avoiding repetition of the subject-matter of other chapters as far as possible.

Etiology. Frequency.—As far back as the history of medicine extends tuberculosis has existed as one of the most frequent of diseases. It affects many different varieties of animals, although to unequal degrees; being most prevalent in man, monkeys, cattle and swine, as well as in poultry, although in the last it is a decidedly different disorder. It is rare in sheep, goats, horses, dogs and cats. Guinea-pigs and rabbits are very susceptible if inoculated. The actual frequency of tuberculous infection, apart from the occurrence of lesions found at autopsy, is difficult to determine. Not all cases which show post-mortem lesions have died of this disease. In many it is only a contributory or an accidental matter. On the other hand, tuberculous infection exists in

many cases which do not come to autopsy. Of 4388 children in the schools of Christiania, as reported by Fröhlich,¹ 2900 were examined clinically and by the cutaneous tuberculin reaction. The former revealed evidences of tuberculosis in 61.5 per cent., and the latter in 83.8 per cent. The influence of the age of the subject upon the frequency of the disease will be considered later (p. 540).

Predisposing Causes.—Much discussed in this connection is the influence of heredity. While direct inheritance of the germ is rare, that of the disposition of the tissues to become tuberculous is very common. Any debilitated condition of the parents increases the susceptibility in the offspring, but the existence of parental tuberculosis is by far the most powerful factor. The development of the disease by association with tuberculous parents and not on account of an inherited tendency is to be carefully excluded in drawing conclusions. Race exerts some etiological influence, but the effects attributed to it depend upon other associated conditions. The Indians, Irish and Negroes appear especially predisposed in the United States. Climate, locality and altitude are prominent factors. With some exceptions damp regions predispose, and dry, elevated localities are unfavorable to the development of the disease. The frequency of tuberculosis in children on the continent of Europe would appear from statistics to be much greater than in the United States, and in certain cities the number of instances of infection developing in early life seems to be particularly high. Unhygienic conditions in general are of great importance. The crowding of children in dirty buildings, the lack of fresh air and sunlight, insufficient nourishment, and the like, are active causes. Previously impaired health also predisposes, especially derangements of the respiratory mucous membrane, and, to a less extent, those of the alimentary tract. Many other diseases predispose, particularly to be mentioned here being measles, pertussis, grippe, repeated attacks of bronchitis, and the existence of hypertrophied tonsils and of adenoid growths. These various agents may act either by rendering the subject liable to the entrance and development of the germs, or by decreasing the local or general resistance of the organism and thus allowing an infection already present in the body in a quiescent condition to assume an active form.

Age exerts a very positive influence. Congenital tuberculosis is occasionally seen, as was proven by Schmorl and Birch-Hirschfeld² and others, and a considerable number of cases have been reported (Pehu and Chalié).³ The occurrence is very possibly more common than generally supposed. The subject has been reviewed by Weber⁴ and by Grullee and Harmes.⁵ Tuberculosis is, however, exceptional in the first 3 months of life, but increases rapidly in frequency of occurrence after this period. Cornet⁶ gives the following figures based on 1542 autopsies on children under 5 years of age:

¹ Norsk. Mag. f. Laegevidensk., 1914, LXXV, 137. Ref. Monatsschr. f. Kinderh. Ref., 1915, XIV, 379.

² Beiträge z. path. Anat. u. z. allg. Path., 1891, IX, 429.

³ Arch. de méd. des enf., 1908, XI, 1.

⁴ Brit. Jour. Child. Dis., 1916, XLII, 321; 359.

⁵ Amer. Jour. Dis. Child., 1915, IX, 322.

⁶ Nothnagel's Encycl. Pract. Med., Amer. Ed., Tuberculosis, 351.

TABLE 74.—INCIDENCE OF TUBERCULOSIS AS DERIVED FROM AUTOPSIES

	Autopsies	Tuberculous	Percentage
Birth to 2 mos.....	486	0	0
2 to 3 mos.....	33	2	6.06
3 to 6 mos.....	76	8	10.53
6 to 9 mos.....	88	15	17.05
9 to 12 mos.....	65	18	27.69
Total under 1 yr.....	748	43	5.75
1 to 2 yrs.....	311	83	26.69
2 to 3 yrs.....	189	56	29.63
3 to 4 yrs.....	160	51	31.86
4 to 5 yrs.....	134	30	22.39
Total.....	1542	263	17.5

There exists, however, considerable variation in many of the published statistics, as may be seen in the following selected examples: In 752 autopsies in children reported by Feldmann¹ tuberculosis was present in 19.6 per cent.;—under 3 months, 6.3 per cent. of all autopsies at this age; 3 to 6 months, 22.8 per cent.; 7 to 9 months, 29.1 per cent.; 10 to 12 months, 22.4 per cent.; 1 to 2 years, 43.1 per cent.; over 2 years, 71.1 per cent. Schlossmann² gives 6.8 per cent. tuberculous in 532 autopsies in infants under 1 year. According to Hamburger³ of 848 autopsies in children in Vienna, 39.53 per cent. showed tuberculosis. In the first 3 months it was present in 4 per cent.; 4 to 6 months, 18 per cent.; 7 to 12 months, 23 per cent.; in the 2d year, 40 per cent.; 3 to 4 years, 60 per cent.; 5 to 6 years, 56 per cent.; 7 to 10 years, 63 per cent.; 11 to 14 years, 70 per cent. The percentages in the records of the Children's Hospital of Philadelphia (Hand)⁴ are also high; 115 of 332 autopsies, or 34.6 per cent., revealing tuberculous lesions, $\frac{1}{2}$ of these cases being in subjects under 2 years of age. Harbitz⁵ for Norway found tuberculous lesions in 40 per cent. of 484 autopsies in children from 1 to 15 years of age. The Paris mortality is shown by the statistics of Comby⁶ upon 1675 autopsies with 638 (38 per cent.) tuberculous. The division according to age was: Of autopsies under 3 months, tuberculous 1.82 per cent.; 3 to 6 months, 18.18 per cent.; 6 to 12 months, 26.25 per cent.; 1 to 2 years, 40.72 per cent.; 2 to 5 years, 60 per cent.; 5 to 10 years, 67.15 per cent.; 10 to 15 years, 71.23 per cent.

The figures as given represent, however, only the finding of tuberculous lesions at autopsy. They do not indicate that the death was due to this disease, nor do they show how often children of different ages, who do not come to autopsy, are subjects of tuberculous infection. In fact, the older the child, the oftener is the tuberculosis lesion a latent or healed process. The question of the frequency of tuberculosis at different periods as shown by the tuberculin-test (see p. 560) has been carefully studied in many quarters. The figures obtained vary somewhat with the locality, as may be seen in the following table:

¹ Budapesti orvosi ujsay, 1906. Ref., Jahrb. f. Kinderh., 1906, LXIV, 763.

² Beiträge z. Klin. d. Tuberc., 1906, VI, 229.

³ Wien. klin. Wochenschr., 1907, XX, 1668.

⁴ Arch. of Pediat., 1903, XX, 247.

⁵ Norsk. Mag. f. Lægevidensk., 1913, 1. Ref. Monatsschr. f. Kinderh., Referat., 1914, XIII, 429.

⁶ XVII Intern. Cong. of Med., 1913, Sect. X, 38.

TABLE 75.—INFLUENCE OF LOCALITY ON AGE-INCIDENCE OF TUBERCULOSIS

Lapage, ¹ Manchester			Veeder and Johnson, ² St. Louis		
Age	Number of cases	Positive reactions, per cent.	Age	Number of cases	Positive reactions, per cent.
0-2 yrs.....	103	32.0	Under 1 yr.....	202	1.5
2-5 yrs.....	209	51.2	1-2 yrs.....	109	5.5
5-10 yrs.....	446	60.6	2-4 yrs.....	163	19.0
10-14 yrs.....	242	60.8	4-6 yrs.....	172	23.0
			6-8 yrs.....	152	29.0
			8-10 yrs.....	126	30.0
			10-12 yrs.....	107	34.0
			12-14 yrs.....	94	38.0
Total.....	1000	55.7	Total.....	1125	21.0

Hamburger and Monti, ³ Vienna			M'Neill, ⁴ Edinburgh		
Age	Number of cases	Positive reactions, per cent.	Age	Number of cases	Positive reactions, per cent.
Under 1 yr.....	23	0.0	Under 1 yr.....	64	14.1
1-2 yrs.....	46	9.0	1-2 yrs.....	61	29.9
2-3 yrs.....	56	20.0	3-4 yrs.....	75	46.6
3-4 yrs.....	75	32.0	5-6 yrs.....	52	28.8
4-5 yrs.....	50	52.0	7-10 yrs.....	79	51.9
5-6 yrs.....	63	51.0	11-14 yrs.....	40	55.0
6-7 yrs.....	46	61.0			
7-8 yrs.....	30	73.0			
8-9 yrs.....	35	71.0			
9-10 yrs.....	26	85.0			
10-11 yrs.....	29	93.0			
11-12 yrs.....	19	95.0			
12-13 yrs.....	17	94.0			
13-14 yrs.....	17	94.0			

The cases of Lapage showed in some instances clinical evidences of tuberculosis; those of Veeder and Johnson and of Hamburger and Monti did not; those of M'Neill were unselected. The cutaneous test was the one employed in nearly all instances; occasionally the intracutaneous method was used.

The obtaining of a positive tuberculin-reaction is not a proof that the clinical manifestations which the child may exhibit necessarily depend upon tuberculosis, but only that this disease exists somewhere in the system, perhaps as a small, entirely inactive focus. It is also probable that children of the better classes, and not examined when suffering from any symptoms, would not give a positive reaction so often. However this may be, the frequency with which tuberculous lesions are found in children dying from other disorders, and the large number of cases in which a positive tuberculin-reaction is obtained in those apparently without tuberculous disease, appears to favor the view of Schlossmann⁵

¹ Brit. Jour. Child. Dis., 1912, 1X, 493.

² Amer. Jour. Dis. Child., 1915, 1X, 478.

³ Münch. med. Woch., 1909, LVI, 449.

⁴ Edinburgh Med. Journ., 1912, I, 324.

⁵ Münch. med. Woch., 1909, LVI, 398.

and of Hamburger¹ that tuberculosis is to a large extent a children's disease, acquired in childhood and often recovered from at this time; although it may in other cases lie dormant and break out actively in adult life.

Exciting Cause.—That the disease is an infectious one was believed from early times, but was first clearly proven by Villemin in 1865.² That it was dependent upon the tubercle bacillus was first demonstrated by Koch in 1882.³ Most authors agree that there appear to be two varieties of the bacillus capable of producing the disease in man: the bovine and the human. The relationship and relative importance of these is still a subject of much discussion. It appears to be proven that the human bacillus can occasionally cause tuberculosis in cattle. In like manner, the bovine germ can certainly produce the disease in man, but the large majority of cases owe their origin to the human variety. This would seem, at least, to be the most frequent experience, although certain investigators, notably of Scotland, among them Mitchell,⁴ Fraser,⁵ and others, insist on the etiological importance of the bovine bacillus especially in children, and chiefly in the production of tuberculosis of the cervical and mesenteric glands and of the bones and joints. There would appear to be a difference in the prevalence of the two forms of the bacillus in different countries, since in the United States, at least, infection by the bovine bacillus is very much less frequent. Park and Krumwiede's⁶ statistics show that tuberculosis of the lungs, meninges and osseous system in children is nearly always of human origin, but that the disease in the intestines and the cervical glands is not infrequently caused by the bovine type; and that there is a greater tendency for bovine tuberculosis to develop in children than in adults. Their statistics are based upon their own and collected reports of cases, and equal 1511 examinations in all, or 545 in subjects up to 16 years of age. The following table is taken from their publication.

TABLE 76.—INCIDENCE OF HUMAN AND BOVINE TUBERCULOSIS, RESPECTIVELY

Diagnosis	Children 5 to 16 years		Children under 5 years	
	Human	Bovine	Human	Bovine
Pulmonary tuberculosis.....	14	..	35	1
Tuberculous adenitis (axillary or inguinal).....	4	..	2	..
Tuberculous adenitis (cervical).....	36	22	15	24
Abdominal tuberculosis.....	8	9	10	14
Generalized tuberculosis of alimentary origin....	3	4	17	15
Generalized tuberculosis.....	5	1	74	7
Generalized tuberculosis including meningitis; alimentary origin.....	1	..	5	10
General tuberculosis including meningitis.....	10	..	76	1
Tuberculous meningitis.....	3	..	28	4
Tuberculosis of the bones and joints.....	41	3	27	..
Genito-urinary tuberculosis.....	2
Tuberculosis of the skin.....	4	6	2	..
Miscellaneous.....	..	1	1	..

¹ Münch. med. Woch., 1908, LV, 2702.

² Gaz. hebdom., 1865, II, 795.

³ Berlin. klin. Woch., 1882, XIX, 221.

⁴ Edinburgh Med. Journ., 1914, XIII, 209.

⁵ Journ. Exper. Med., 1912, XVI, 432.

⁶ Journ. Med. Res., 1912-13, XXVII, 109.

The germs are found in tuberculous lesions wherever situated and in the secretions and excretions from the affected tissues, particularly when the process is active. Outside of the body they are widely diffused and very abundant, the chief source being the sputum, in which they are present in enormous numbers. They are capable of living and remaining virulent for weeks in a dried state in the dust from rooms occupied by consumptive patients (Cornet),¹ but are killed by a temperature of 60°C. (140°F.) continued for from 15 to 20 minutes. The general opinion is that they are seldom to be found in the blood.

Mode of Transmission and Portal of Entry.—Except for the rare cases of congenital tuberculosis in which the infection is through the blood, and the exceptional cases of tuberculosis by direct inoculation, as by ritual circumcision or through other lesions of the skin, the germ enters the body either by the respiratory or the alimentary tract. When infection is by the inspired air, the tubercle bacilli, often attached to particles of dust or to fine, moist drops expelled by the coughing of some patient, are deposited upon the mucous membrane of the nasopharynx, larynx, trachea, bronchi or lungs. If any catarrhal or other lesion exists the penetration of this membrane is rendered much easier, but it can take place without a lesion being present. It has been believed that a tuberculous infection either may develop at the site of invasion, or that the germs may pass through the mucous membrane without injury to it. It is probable, however, as maintained by Parrot,² Küss,³ Albrecht,⁴ Ghon⁵ and others, that the primary tuberculous lesion is always situated at the portal of entry, although it may be small and undiscovered except by the most careful search. This appears to be the present prevailing opinion. The lesion here suffers the usual changes of caseation, calcification or softening, and from this focus the bacilli travel by way of the lymphatic vessels to the regional lymphatic glands, usually the tracheobronchial. These act as sieves and may arrest the further progress of the germs, which can lie dormant here for an indefinite time, as shown by the investigations of Loomis,⁶ Pizzini,⁷ Beitzke⁸ and others, exerting no influence whatever on the general health, although the condition is an ever-present menace. Infection of the organs, or of the system at large, is the result of the inflammatory process increasing in the primary focus or in the lymphatic glands, and the bacilli extending thence by way of the lymphatics; or by the process finally involving the blood-vessels, and the germs then entering the blood-current.

When the infection is by the alimentary tract the bacilli enter from the pharynx, tonsils, esophagus, stomach, or intestine, being attached to the food, or obtained from toys and the like, or even from the infant's fingers. As with the respiratory tract there is usually a tuberculous lesion, although small, at the portal of entry. When the intestine is the seat of the primary lesion, the bacilli pass thence into the mesenteric glands and finally to other parts; when entrance is by way of the tonsillar tissue the cervical glands generally arrest further extension of the disease.

Whether infection by the respiratory or the alimentary tract is the

¹ Zeit. f. Hyg., 1888, V, 191.

² Compt. rend. soc. de biol. de Paris, 1876, III, 308.

³ De l'hérédité parasitaire de la tuberculose humaine, Paris, 1898. Ref. Ghon.

⁴ Wien. klin. Wochenschr., 1909, XXI, 327.

⁵ Die primäre Lungenherd bei der Tuberculose der Kinder, Berlin, 1912.

⁶ Med. Rec., 1890, XXXVIII, 689.

⁷ Zeitschr. f. klin. Med., 1892, XXI, 329.

⁸ Virchow's Archiv., 1912, CCX, 173.

more frequent in children has been, and still is, a subject of widespread discussion. Except in the British statistics there is little question, as post-mortem examination has repeatedly shown, that primary lesions are much more often found in the region of the respiratory tract, including the tracheobronchial lymph-glands (see p. 556), and a logical conclusion would appear to be that the gland-filter situated nearest to the portal of entry will be the one first affected. On the other hand, it has been claimed by those who maintain that infection by way of the intestine is the more common, that the bacilli are not filtered out by the mesenteric glands, but are carried to the general circulation by the thoracic duct and finally are deposited in the lungs and the tracheobronchial glands, which are the parts most susceptible to the disease in early life; and that consequently apparently primary lesions of these glands do not exclude the possibility of alimentary infection.

Even experimental evidence is contradictory. Thus, as illustrating the opposing views, Straus¹ showed that tubercle bacilli injected into the stomach readily cause a general tuberculosis, and Calmette, Guérin and Délearde² that respiratory involvement could be produced in this way without any affection of the mesenteric glands. On the other hand Cornet,³ Pfeiffer and Friedberger,⁴ and Findlay⁵ demonstrated the great ease with which respiratory tuberculosis could be brought about by inhalation experiments, and that injection of germs into the stomach produced tuberculosis with difficulty.

Directly bearing upon this matter is the question of the danger of milk from tuberculous cattle. That this exists has been practically denied by Koch,⁶ but affirmed by von Behring⁷ and other investigators. The preponderance of the evidence at the present time seems to indicate that infection with the bovine bacillus contained in milk from tuberculous cattle is a possibility, but comparatively infrequent; and that the danger is much over-rated. Cattle which react to tuberculin but reveal no tuberculous foci usually have no bacilli in their milk. The same is true of infection by the human bacillus in the milk of tuberculous mothers. More is to be feared from the contamination of cows' milk by the *human* bacilli which may enter it from the air, just as they may contaminate any other article which enters the infant's mouth. At the same time it is unwise to employ milk from tuberculous cattle; and whether or not the milk from a tuberculous mother is dangerous, the intimate association of the infant with her certainly is so, and should be prevented. As regards the flesh of tuberculous cattle, infection by it is perhaps possible, but certainly unlikely.

Pathological Anatomy. The Histology of Tubercle.—The basis of the pathological changes of tuberculosis is the miliary tubercle, which is produced by the irritation of the rapidly multiplying bacilli. It consists of a grey, translucent body about $\frac{1}{20}$ to $\frac{1}{5}$ inch (0.127 to 0.5 cm.) in diameter surrounded by a reticulum of connective tissue, and composed of lymphoid and epithelioid cells the result of proliferation of the tissue cells, and of leucocytes, at first polymorphonuclear, later chiefly mono-

¹ Arch. méd. exper., 1896, VIII, 689.

² Compt. rend. acad. sci., CXLII, No. 21. Ref. Zentralbl. f. inn. Med., 1906, XXVII, 1090.

³ Nothnagel's Encyclop. Pract. Med. Tuberc., 98; 143.

⁴ Deutsch. med. Wochenschr., 1907, XXXIII, 1577.

⁵ Zeit. f. Kinderh., Orig., 1913, VIII, 503.

⁶ Brit. Med. Journ., 1902, II, 1885.

⁷ Deutsch. med. Wochenschr., 1903, XXIX, 689.

nuclear, migrating from the neighboring blood-vessels. Bacilli are present in some of the epithelioid cells. In some of the tubercles giant cells are also found, their number being inversely proportional to the number of bacilli present. The tubercles may remain scattered and mostly separated from each other, or may be grouped in large masses. As growth progresses degenerative changes take place. Caseation is the most common, the tubercle becoming yellow, containing many bacilli, and finally softening. This is followed by an inflammation of the surrounding tissue and the formation of pus through a mixed infection. Less frequently calcification occurs, seen oftenest in the lymphatic glands. Sometimes, especially if the tubercles remain discrete and miliary, sclerosis takes place, a firm fibrous structure resulting.

Regions Oftenest found Affected by Tuberculosis at Different Periods of Infancy and Childhood.—*Age* is a prominent factor in this connection. It is to be borne in mind that the process is seldom limited to any one locality, and that the region most frequently attacked does not necessarily produce the most important symptoms. In fact, tuberculosis may be only a post-mortem finding, death being dependent upon some other disease. Taking infancy and childhood as a whole, the statistics compiled by Freeman¹ in 2288 autopsies in tuberculous children reported by different observers show involvement of the lungs in from 71 to 100 per cent.; of the bronchial lymph-nodes in from 76 to 99 per cent.; of the intestines in from 23 to 55 per cent.; and of the mesenteric lymph-nodes in from 16 to 40 per cent.

In the *first 2 years of life* the regions oftenest found diseased are the bronchial lymphatic glands, lungs, and pleura. The tubercles may be scattered over the surface of the lung or may involve a small part of its parenchyma. Most frequently there develop the lesions of acute or subacute tuberculous bronchopneumonia, and less often those of miliary tuberculosis of the entire lung. There may be numerous small cavities; but the formation of large cavities, as seen in phthisis, is less common at this period. The bronchial glands are more or less enlarged and cheesy, and not infrequently suppurate. Sometimes only a few are affected; sometimes large masses of much enlarged glands are present. Involvement of the mesenteric glands is less frequent than that of the bronchial; that of the cervical glands not as common as later. Tuberculous meningitis is very frequent, seen oftenest in combination with other clinical manifestations, especially those of bronchopneumonia or of a general tuberculosis. Stirnimann² found the latter in 54.4 per cent. of 42 cases in 591 autopsies in the 1st year of life. Tuberculosis of the peritoneum, the intestine, and the bones is not frequent in infancy. At this period of life there is especially to be noted the tendency for tuberculosis to become widespread. Raczyński³ states that in 614 cases of tuberculosis as seen at autopsy, up to 3 years about 50 per cent. had a general tuberculosis.

In *early childhood*, from the 3d to the 5th year inclusive, general tuberculosis and meningitis remain very common, and involvement of the cervical and mesenteric glands, and of the intestine, peritoneum and bones increases in frequency. Tuberculosis of the lungs is, however, probably the most common form, with that of the pleura and bronchial glands nearly as often seen as this.

¹ Med. News, 1905, May 27.

² Jahrb. f. Kinderh., 1906, LXIII, 130.

³ Jahrb. f. Kinderheilk., 1901, LIV, 67.

In *later childhood* tuberculous meningitis, although still frequent, is not so often observed as in earlier childhood. Cervical adenitis and involvement of the bones and joints remain common, while tuberculous peritonitis is increasingly often seen.

The kidneys, liver and spleen may show miliary tubercles on their surface at any age in cases of general miliary tuberculosis. The larynx is seldom involved. The pancreas, thyroid, thymus, heart, and genito-urinary tract are regions only exceptionally attacked in early life. In general, there is a predisposition at this age to involvement of the glands, while in adults the mucous membranes are oftener attacked.

Seat of the Primary Lesions.—This, of course, can be merely surmised in many instances. Reference is not made here to the original lesion at the portal of entry, often small and undiscovered, but to those lesions readily seen and having the appearance of having existed longest, and from which the extension of the process could be considered to have taken place. In 200 autopsies on tuberculous children, reported by Northrup¹ and by Bovaird,² this oldest lesion was in the respiratory tract, including the bronchial glands, in 148, and in the intestines and mesenteric glands in 3. In 119 autopsies on tuberculous children published by Holt³ the oldest lesion was in no instance in the intestinal tract, and in 63 per cent. of the cases there was no intestinal involvement whatever. In 115 autopsies on tuberculous children in the Children's Hospital of Philadelphia, recorded by Hand⁴ apparently primary lesions of the intestine or mesenteric glands were found in 10. In 335 autopsies on children with tuberculous lesions reported by Hamburger⁵ there was no certain case of primary intestinal or mesenteric tuberculosis. Albrecht⁶ in 1060 autopsies showing tuberculosis in children found only 0.66 per cent. exhibiting undoubted primary intestinal tuberculosis. All these figures indicate the great preponderance of lesions in the respiratory tract as generally observed.

On the other hand, British statistics show somewhat different figures. J. W. Carr⁷ found in 120 autopsies 79 with primary respiratory lesions, and 20 with primary intestinal or mesenteric glandular involvement; and Still⁸ in 269 cases noted the primary localization as follows: Lungs 138; intestines 63; bones and joints 5; ear 15; unknown situation 46. The contrast between British and American experience is well illustrated in the collected statistics published by Bovaird⁹ as follows:

TABLE 77.—INTESTINAL TUBERCULOSIS; ENGLISH AND AMERICAN STATISTICS

	Cases	Primary intestinal	Per cent.
English.....	1161	236	20.33
American.....	620	21	3.39

In addition is to be mentioned the occasional occurrence of the primary localization in the tonsils or adenoids, Wood¹⁰ finding tuberculosis

¹ New York Med. Journ., 1891, LIII, 201.

² New York Med. Journ., 1899, LXX, 1.

³ Med. News, 1896, LXIX, 656.

⁴ Arch. of Ped., 1903, XX, 248.

⁵ Wien. klin. Wochenschr., 1907, XX, 1069.

⁶ Wien. klin. Woch., 1909, XXII, 327.

⁷ Lancet, 1894, I, 1177.

⁸ Pediatrics, 1899, VIII, 335.

⁹ Sixth Internat. Cong. Tuberc., 1908, II, 4, 451.

¹⁰ Journ. Amer. Med. Assoc., 1905, XLIV, 1425.

in 5.2 per cent. of 1671 collected cases in which examination was made of the tonsillar tissue removed from individuals apparently free from the disease in other respects. Doubtless these organs are oftener the primary seat than has usually been supposed.

Lesions Oftenest Producing Death.—It by no means follows that the parts oftenest affected are those the disease of which occasions the death of the patient. Thus, in infancy, although pulmonary involvement, usually in the form of bronchopneumonia, is the most frequent cause of death, in many pulmonary cases the fatal issue depends actually upon a general miliary tuberculosis, or, oftener, a tuberculous meningitis. Involvement of the bronchial lymphatic glands, although so frequent, rarely produces fatal symptoms. In early and later childhood meningitis, secondary to involvement of the lungs, bones, or lymph glands, very often occasions the fatal termination. Shennan¹ found this true in 44.5 per cent. of 413 cases of tuberculous disease of various sorts in children. Tuberculous peritonitis is also a not infrequent cause of death in later childhood, while tuberculous pleurisy, although common, is rarely in itself fatal.

Clinical Forms of Tuberculosis.

(A) **GENERAL TUBERCULOSIS.**—This form consists in a widespread development of tubercles secondary to some small caseous area. Through the entrance of large numbers of bacilli into the blood the disease is spread more or less widely throughout the body, the extent varying greatly with the case. Sometimes the various organs are crowded with tubercles, especially well seen in the lungs; sometimes these are much more scattered and fewer. The symptoms and the rapidity of the course of the disease vary accordingly. The liver is oftener affected in children than in adults. The tubercles are commonly larger in the child than in the adult and in the less rapid cases assume a considerable size through confluence, many of them becoming caseous and breaking down. This variety of the disease may exhibit itself in two forms, (1) the *Typhoid* and (2) the *Marantic*.

1. The Typhoid Form; Acute Miliary Tuberculosis. This is seen chiefly in infancy and early childhood. The initial symptoms are very vague, consisting of loss of appetite, debility, loss of weight, and other evidences of a general impairment of health. Fever of an irregular and uncharacteristic type soon develops; debility increases; the tongue

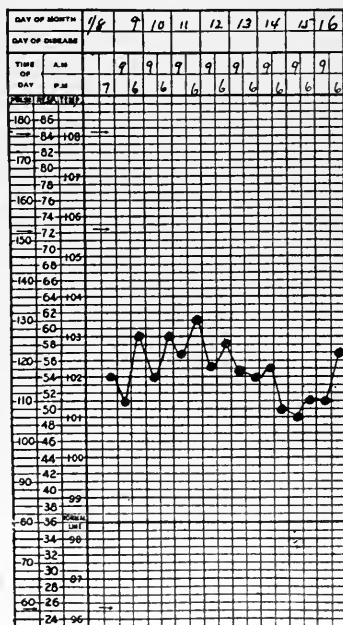


FIG. 173.—GENERAL TUBERCULOSIS, TYPHOID FORM.

George D., aged 5½ years, colored. Jan. 8. In bed for a week with fever and debility. Is apathetic, weak, tongue coated. No physical signs in chest or abdomen. Typhoid fever suspected. Jan. 14, gradually increasing somnolence, slight, varying rigidity of limbs, leucocytosis 7410, negative Widal reaction; Jan. 16, undoubted slight signs of consolidation in right lung. Death. Autopsy showed general miliary tuberculosis.

¹ Sixth Internat. Cong. Tuberc., 1908, II, 4, 367.

becomes dry; the pulse and respiration are accelerated; there is apathy, dullness, sometimes delirium and finally coma. The tympanites and enlarged spleen may, with the continued fever and other symptoms, strongly suggest typhoid fever (Fig. 173). In other cases the temperature is almost too irregular for this disease, and often only slightly or, exceptionally, not at all elevated. No cause for the symptoms can be found. Temporary improvement may occur, although the emaciation

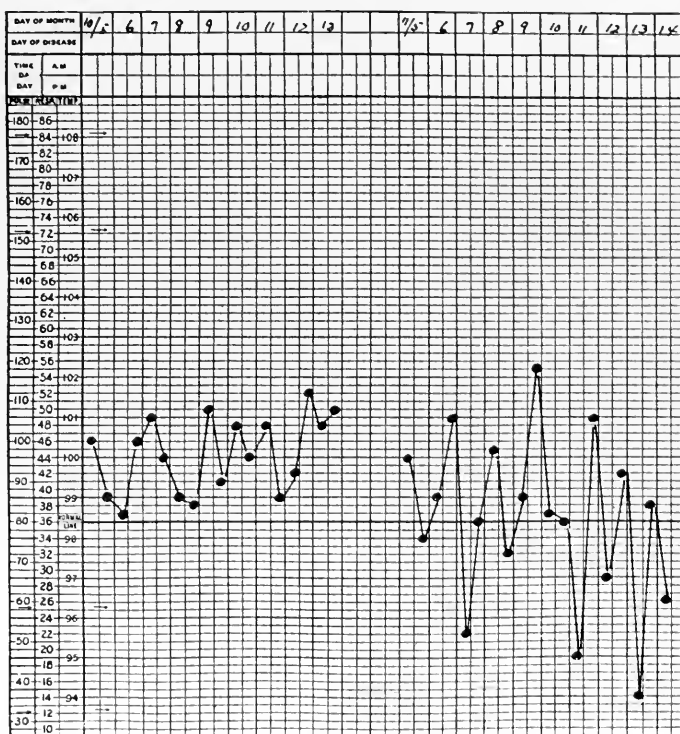


FIG. 174.—GENERAL TUBERCULOSIS, MARANTIC FORM. FINAL BRONCHOPNEUMONIA AND MILIARY TUBERCULOSIS.

Chas. McL., aged 1 year. Said to have been ill for 6 months. Been suffering latterly from fever, loss of weight, slight cough, poor appetite and loose stools. *Examination*.—Greatly emaciated; anemic; enlarged cervical, axillary, inguinal and abdominal glands; possible slight impairment of percussion resonance, and harsh respiration; distended abdomen. Later positive evidence of pulmonary involvement. Had daily a moderate number of soft or liquid undigested stools. Death about 1 month later. *Autopsy*.—Tuberculous bronchopneumonia of left lung and root of right lung; miliary tuberculosis both lungs and of the spleen; tuberculous alteration of intestine; adenitis of mesenteric, retroperitoneal and bronchial glands.

does not disappear nor the strength greatly increase. Finally some localizing symptoms may develop, especially those of pulmonary consolidation or of meningitis; less often of peritonitis. The course of the disease is rapid, death occurring usually in a few weeks. The differential *diagnosis* is extremely difficult. Suggestive of tuberculosis is the more irregular fever, the tendency to rapid respiration, the failure of the fever to lessen at the end of 3 or 4 weeks, and especially the persistent absence of the Widal reaction. Finally the development of localizing symptoms

may make the diagnosis of tuberculosis almost certain; but this occurs generally only a few days before death.

2. The Marantic Form.—Infants not infrequently exhibit a somewhat more chronic form of tuberculosis, closely simulating marasmus. The symptoms are entirely uncharacteristic, progressive wasting and anemia not being the principal ones. There is no fever, or occasional and irregular elevations, and no respiratory or gastrointestinal disturbances sufficient to account for the condition. The disease can in no way be distinguished from other marantic states, unless the case comes to autopsy. In other instances there develops a few weeks before the end of life more or less constant but moderate fever of irregular type, with slight symptoms suggesting bronchopneumonia (Fig. 174). The respiration is somewhat accelerated yet seldom decidedly dyspneic, there is slight cough, and the physical signs in the lungs are usually not well-marked, or only those of bronchitis. Sometimes vomiting or, especially, diarrhea may become troublesome, or the symptoms of meningitis close the scene. Generally, however, death appears to be due to progressive exhaustion. The course of the case, although decidedly more prolonged than in the typhoid form, is brief; at longest a few weeks after continued fever or localizing symptoms appear, but often much longer from the first beginning of signs of illness. The *diagnosis* is always difficult and usually impossible; until, perhaps, shortly before death. Even the development of signs of bronchopneumonia is not conclusive, since non-tuberculous bronchopneumonia is so frequently a terminal condition in infantile atrophy. The principal diagnostic aid is to be sought in the history. The entire absence of discoverable reason for the continued wasting, the absence of evidences of chronic or repeated intestinal autointoxication, and the fact that diarrhea, cough, and vomiting have followed emaciation rather than preceded it, are reasons for suspecting tuberculosis. The tuberculin-test may be of value in some cases.

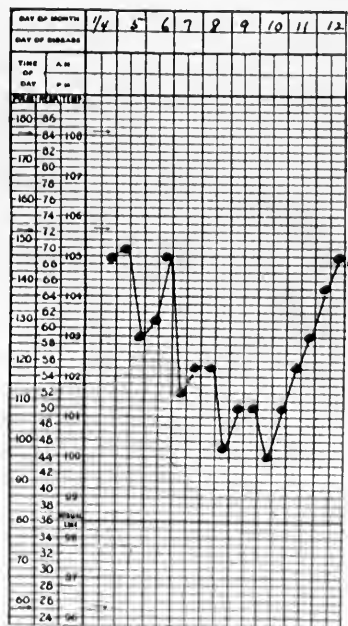


FIG. 175. ACUTE MILARY TUBERCULOSIS OF THE LUNGS.

Bertha D., aged 3 years, admitted to Children's Hospital of Philadelphia, Jan. 4. About Dec. 15, developed cough and fever which continued. *Examination on admission.* Dyspnea, rapid respiration, bases of lungs filled with fine riles, and bronchophony heard. Child toxic; Jan. 11, been growing worse, convulsive movements, unconscious, harsh breathing and numerous scattered riles over both lungs in front and behind, no bronchial respiration or dullness on percussion; Jan. 12, death. *Autopsy.* Lungs crowded with milary tubercles throughout. Moderate enlargement of the tracheobronchial glands. Moderate development of tubercles on surface of liver and spleen and on the surface of the cerebrum.

(B) TUBERCULOSIS OF SPECIAL REGIONS. Here are to be included a large number of forms of tuberculosis in which the disease is confined to, or preponderates in, certain regions or organs of the body. Many of the conditions will be simply mentioned briefly, the fuller description of them being found elsewhere.

1. Tuberculosis of the Lungs.—Several varieties of this may be seen:

(a) *Acute Miliary Tuberculosis of the Lungs* (Fig. 175).—This is the form of the general miliary tuberculosis described in which the localization is most prominent in the lungs from the onset. It is most common after the age of infancy. There is persistent fever of irregular but not hectic type; rapid respiration, which is sometimes dyspneic; prostration; rapid pulse; cough; and sometimes cyanosis. The physical signs in the lungs are often poorly marked and uncharacteristic. Later the evidences of tuberculosis elsewhere may show themselves, the child dying, possibly, from meningitis. The course of the case is short. At autopsy the lungs are found filled with miliary tubercles.

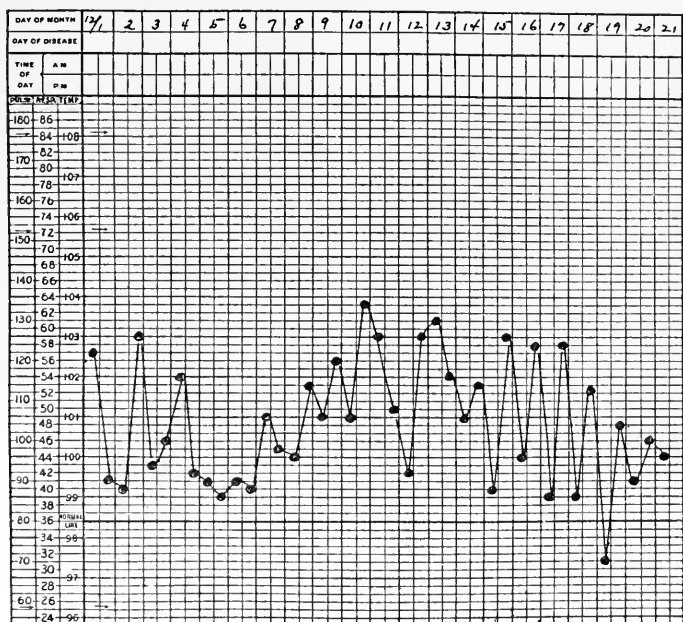


FIG. 176.—TUBERCULOUS BRONCHOPNEUMONIA.

Charles T., aged 1 year. Is said to have been ailing 6 weeks before seen. In the Children's Hospital nearly 1 month. Increasing impairment of percussion resonance and of bronchial respiration over both lungs, with numerous râles. Increasing weakness and loss of weight, petechiæ, cough, anorexia, D'Espine's sign. Tubercle bacilli found in sputum. *Autopsy*.—Bronchial and mesenteric glands much enlarged and tuberculous. Caseous pneumonia of both lungs. Cavity size of walnut in right lower lobe. Miliary tuberculosis of spleen and of pleura.

(b) *Acute Tuberculous Bronchopneumonia*.—This is one of the most frequent manifestations of tuberculosis in children. It is seen oftenest in infancy and especially, in early childhood; may be primary in the lungs or secondary to tuberculosis in some other part of the body, such as the bones, pleura, peritoneum, and particularly the bronchial lymphatic glands; or may follow some other disease, especially pertussis, grippe, measles, or even bronchitis or simple bronchopneumonia. The pathological lesions are the same as those of non-tuberculous bronchopneumonia, with the addition of the presence of tubercle bacilli and the development of tubercles and of the degenerative changes which subsequently take place in these, and which result in the formation of many

smaller and larger caseous areas, and often finally of cavities, if life continues a sufficient time; generally small and centrally located, but sometimes of considerable size. I have seen a cavity as large as an average orange, occupying the entire upper right lobe in a child of 7 months. Occasionally rupture into the pleural sac takes place. The large, slowly developing, encapsulated cavities characteristic of phthisis in the adult are usually absent in childhood until the age of puberty is approached. With the tuberculous process in the lungs is always associated similar disease of the bronchial lymph-nodes. The clinical picture does not differ materially from that of simple bronchopneumonia. (See Bronchopneu-

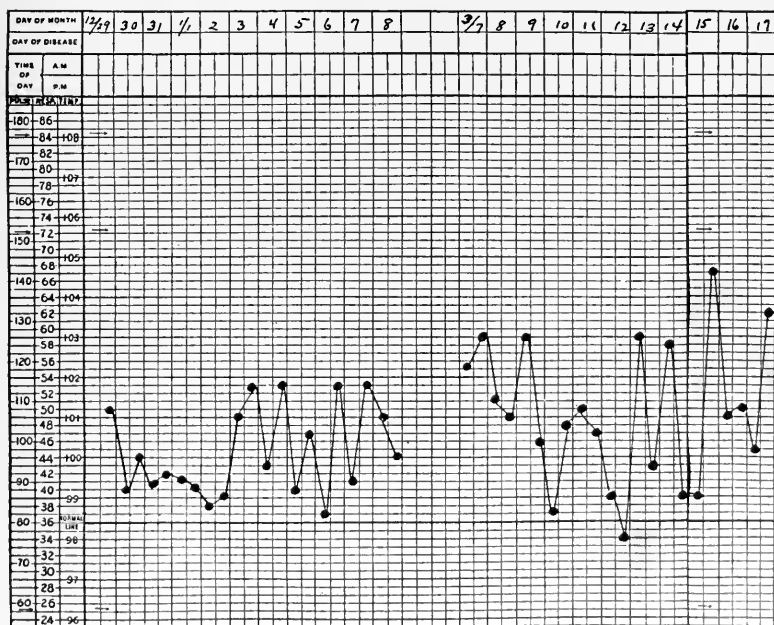


FIG. 177.—SUBACUTE TUBERCULOUS BRONCHOPNEUMONIA WITH CAVITY-FORMATION. Chart shows the first and last 11 days of observation in hospital.

William K., aged 11 months when admitted to the Children's Hospital, Dec. 26, 1916. Died Mar. 18, 1917, aged 14 months. Cough and debility since age of 3 months. While under observation considerable dulness on percussion throughout the right lung; many crackling râles and feeble breath-sounds, not bronchial. Before death there developed bronchial respiration over the upper part of the right lung; distinctly cavernous at the apex. *Autopsy* showed enlarged bronchial glands especially on the right side; the right pleura firmly adherent; right lung tuberculous throughout, a cavity the size of an orange occupying the whole of the upper lobe.

monia, p. 58.) Very frequently, however, the onset is more gradual, the principal early symptoms being cough, loss of weight, debility, increased rapidity of respiration and pulse-rate, and moderate fever of an irregular type (Fig. 176). Later dyspnea becomes decided; the cough worse; the temperature higher, and there is cyanosis and increasing weakness. The physical signs are the same as those of simple bronchopneumonia, the lesions usually being scattered to a varying extent throughout both lungs, although most apparent in one. The pseudolobar form is less often seen. When localization occurs it is oftener in the upper lobe and toward the hilus than is the case with simple bronchopneumonia. Yet there is

such variation possible in the symptoms and physical signs that the *diagnosis* of the tuberculous nature of the case is often impossible. Suggestive of tuberculosis is the development of bronchopneumonia during convalescence from measles or pertussis, or after a period of wasting and ill-health such as occurs in the general tuberculosis of infants; the discovery of tuberculosis elsewhere in the body; the failure of conva-

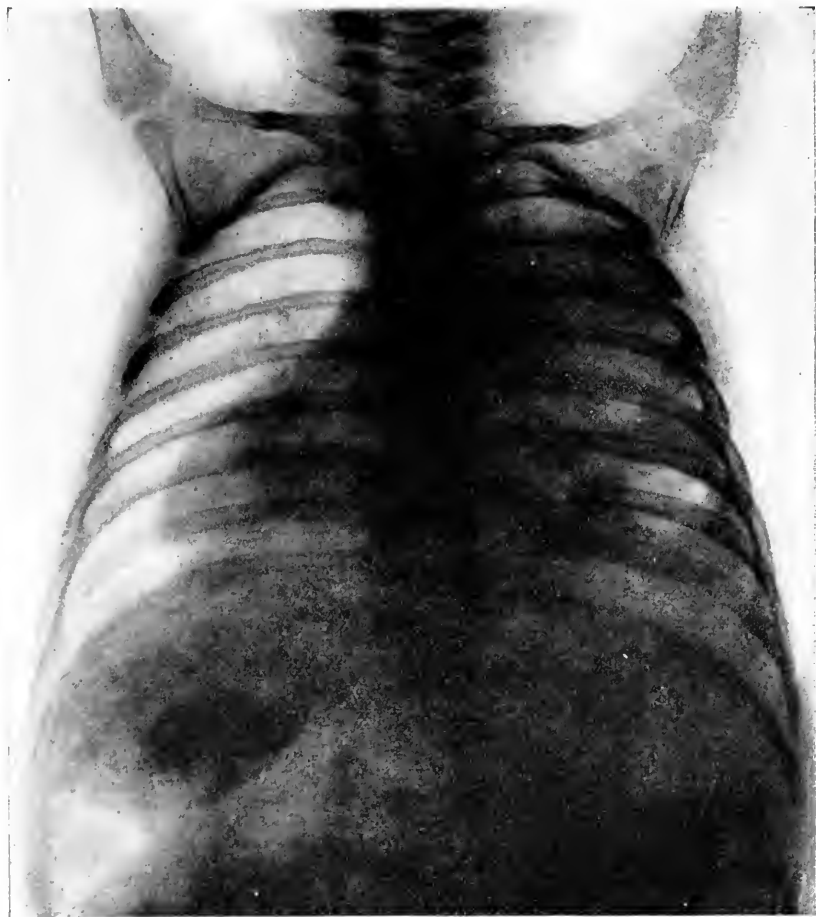


FIG. 178.—RADIOGRAPH OF TUBERCULOUS BRONCHOPNEUMONIA.

Same case as in Fig. 177. Shows advanced lesions of tuberculous bronchopneumonia in the right lung and to a less extent in the left. Viewed from behind.

lescence to begin at the time which may reasonably be expected in simple bronchopneumonia; and the consequently longer course, continuing perhaps for several weeks. Tubercle bacilli may often be found in the secretion from the lungs (see p. 560) or the tuberculin-reaction may be obtained. In rare instances hemoptysis may occur. I have, however, seen a profuse hemorrhage fatal in a few minutes in an infant of 14 months.

(c) *Subacute and Chronic Pulmonary Tuberculosis*.—This may show itself in several forms in children.

(α) *Subacute and Chronic Tuberculous Bronchopneumonia* (Figs. 177-180).—The subacute type of this condition constitutes merely a protracted form of acute tuberculous bronchopneumonia and is directly continuous with it. It may last for some months and terminate fatally, or may, less often, pass into the chronic form. The chronic form may also develop without being preceded by any well-marked attack of bronchopneumonia. In some cases there may have been only attacks of what was supposed to be a bronchitis. Whatever the mode of onset,

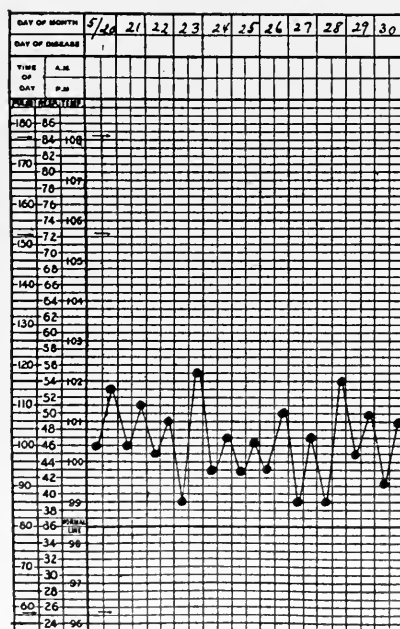


FIG. 179.—CHRONIC TUBERCULOUS BRONCHOPNEUMONIA.

George T., 2½ years old. Taken ill in summer of 1916 with general poor health. In the autumn symptoms of ileocolitis. Recovered from this, but never regained strength. Had dry cough, sometimes troublesome; accelerated pulse and respiration; irregular fever, the last certainly from December. First seen Apr. 4, 1917. Under observation was emaciated, good appetite, bowel movements generally normal, slight cough, nasal obstruction with adenoids, respiration 30 to 40, fretful, debilitated, fever daily, positive von Pirquet, no tubercle bacilli found in secretion obtained from throat, tuberculin under chin, enlarged glands each side of neck. Repeated physical examinations of chest showed nothing abnormal, except deficient expansion; but x-ray examination (Fig. 180) revealed numerous scattered lesions. Went to seashore May 30, 1917, with condition unchanged, and died July 24.

the recovery is not complete and the child is left with debility and cough. Repeated exacerbations occur at intervals and gradually decided physical signs of chronic bronchopneumonia develop, if not present previously. (See Chronic Bronchopneumonia, Vol. II, p. 90.) Exceptionally the disease may begin insidiously, with wasting, continued fever, and more or less cough. The lesions may not be discoverable, or only unsatisfactorily so by physical examination; but the x-ray may show numerous scattered areas (Fig. 180). The case may continue for months and terminate finally by exhaustion or through the development of some acute tuberculous process.

(β) *Hilus Tuberculosis*.—This is a condition arising in connection with tuberculosis of the tracheobronchial glands, especially those adjacent to the hilus of the lung. The lesion may be primary at this portion of the lung, and in any event the glands are affected secondarily. It may develop at any period of early life, even infancy. The onset is insidious and the symptoms vague, consisting of malaise, diminished appetite, debility, and possibly evening rise of temperature. There is a positive tuberculin-reaction, but an absence of the ordinary physical signs. The

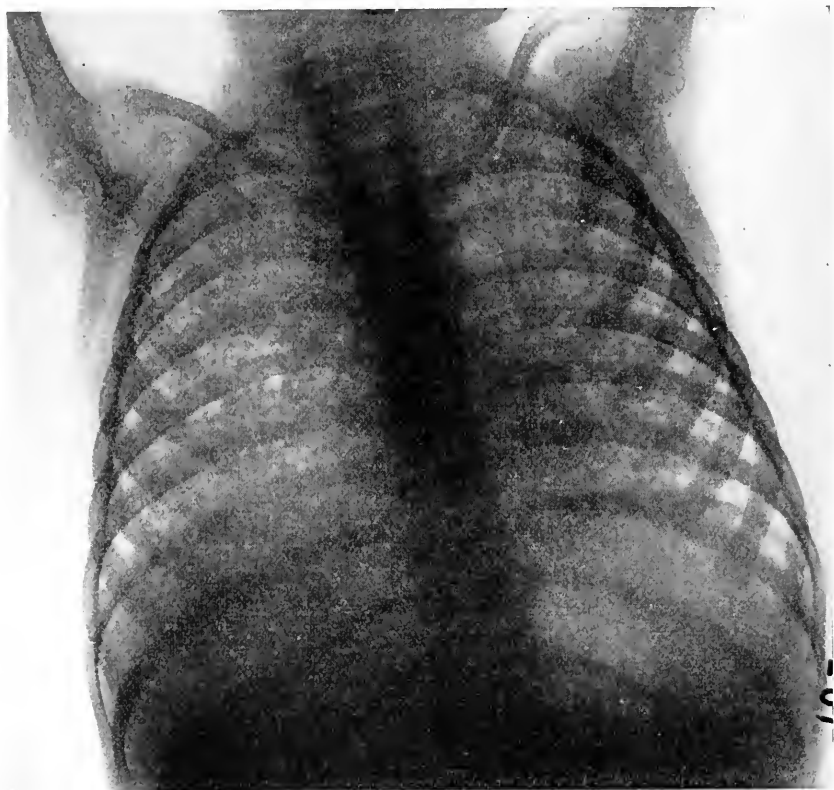


FIG. 180.—RADIOGRAPH OF CHRONIC TUBERCULOUS BRONCHOPNEUMONIA.

Same case as Fig. 179. Shows widespread infiltration with numerous scattered lesions.

involvement of the pulmonary tissue of the hilus cannot be distinguished by physical examination from that of the glands of the hilus which accompanies it. The x-ray examination may show fine lines radiating from the root of the lung (Fig. 181), indicating probably an involvement of the pulmonary parenchyma of the hilus-region (Stoll and Heublein).¹ The condition runs the chronic course of tuberculosis of the tracheo-bronchial glands.

(γ) *Primary Pulmonary Foci of Ghon*.—The name of Ghon² is commonly associated with these lesions, although they were earlier described by others. They consist of small, round foci from that of a millet seed

¹ Amer. Jour. Med. Sci., 1914, CXLVIII, 382.

² Der primäre Lungenherd bei der Tuberkulose der Kinder, 1912.

to that of a hazelnut or even larger in size (Albrecht).¹ They are generally single or but few in number and are situated in different parts of one or both lungs. The lesions represent the seat of primary pulmonary involvement. Any portion of the lung may be attacked, although there is a somewhat greater disposition to involve the upper lobe. They may occur at any period of early life, but with diminishing frequency as infancy is past. The lesions may become caseous, calcified, or shrunken,

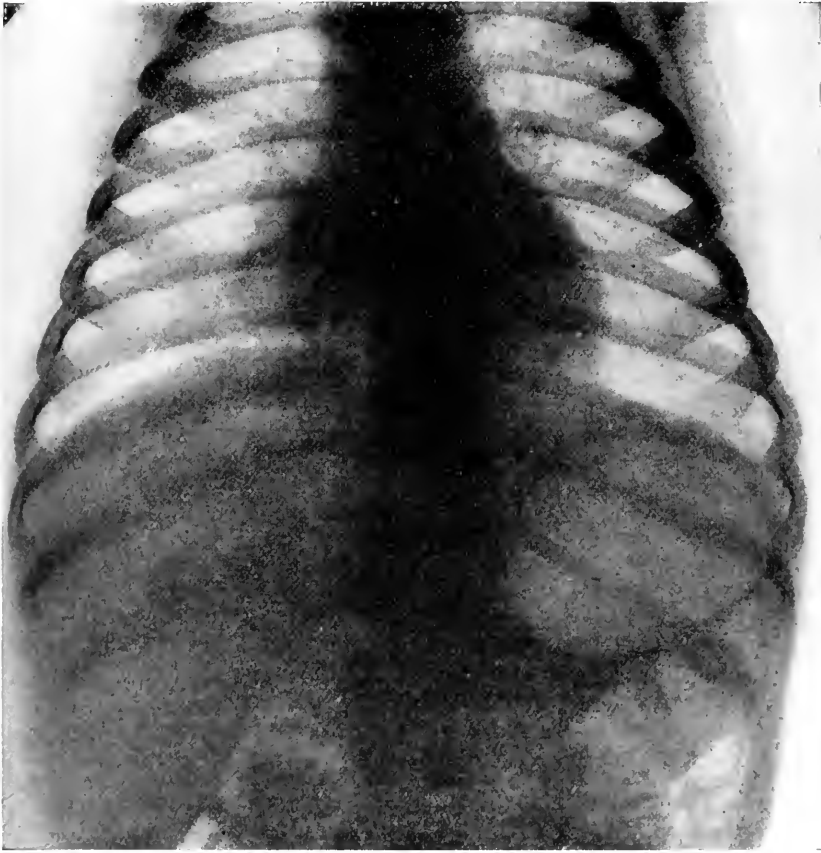


FIG. 181.—RADIOGRAPH OF TUBERCULOSIS OF THE BRONCHIAL GLANDS AND OF THE HILUS OF THE LUNGS.

The ray-like shadows indicate the infiltration along the course of the bronchial tubes.

and may continue for an indefinite time without the production of symptoms, although the neighboring lymphatic glands are always secondarily involved. The condition in this form is of importance only as indicating the primary source of the extension of tuberculosis which later sometimes takes place.

Following another course, and with increasing frequency in proportion to the youth of the patient, the foci instead of becoming dormant remain moderately active, the child exhibiting irregular or no fever, debility, loss of weight and a chronic cough; physical signs being

¹ *Wien. klin. Wochenschr.*, 1909, XXII, 327.

absent except perhaps some scattered râles. The tuberculin-reaction is positive and in some cases tubercle-bacilli may be found in the sputum. The lesions at autopsy consist of the few foci described, which perhaps have become broken down and formed cavities; the combined involvement of the tracheo-bronchial glands; and the evidences of fresh tuberculous processes in the neighborhood of the foci or elsewhere. The prognosis in these more active cases is uncertain. The condition is liable to eventuate in an acute tuberculous bronchopneumonia or a more widespread tuberculosis, but especially in tuberculous meningitis.

(*δ*) *Phthisis*.—This is the form of the disease so common in adult life. It is rarely found in infancy and early childhood, and even in later childhood it is very much less frequent than after this period. Statistics vary considerably, but it is interesting to note that Sawyer¹ in a physical examination of 8000 children under 15 years of age found only 15 in which a diagnosis of phthisis could be made with reasonable certainty. Its symptoms, physical signs and treatment are much the same as those of phthisis in adult life. Only to be noted here is the lesser frequency of cough, involvement of the larynx, dyspnea, and of hemoptysis. The disease is to be distinguished chiefly from chronic bronchiectasis, and from those cases of chronic simple bronchopneumonia in which the occurrence of consolidation about a large bronchus strongly suggests the presence of a cavity. Abscess of the lung, too, may closely simulate it. The obtaining of the tubercle-bacilli from the sputum or the presence of the tuberculin-reaction may settle the diagnosis. The infrequency with which cavity-formation takes place in the cases in children denominated phthisis by many observers raises the question whether, strictly speaking, many of those included in statistics should not be considered a chronic disseminated tuberculous bronchopneumonia, rather than phthisis as it occurs in adult life.

2. Tuberculosis of the Lymphatic Glands. (*Scrofula, Tabes Mesenterica, etc.*).—The involvement of the lymph-nodes is one of the most common forms of tuberculosis in early life, the frequency of its occurrence being much greater than in adults. That of the internal glands is often combined with evident tuberculosis of other parts, the symptoms of which give the clinical evidences of tuberculous infection.

(*a*) *Tuberculosis of the Tracheo-bronchial Lymphatic Glands* (Fig. 181).—These glands include those about the trachea and the main bronchi and those following the major subdivisions of the bronchus at and beyond the hilus of the lungs. The process may be distinctly secondary to lesions in the lungs; or apparently primary, although, in reality, dependent upon some small undiscovered pulmonary tuberculous focus. There are, as a rule, few if any clinical manifestations, and the condition is distinguished only post-mortem. This is especially true of infancy, but even after this period the clinical picture is generally indefinite. In some cases distinct evidences of pressure or of rupture of a suppurating gland become manifest. The symptoms will be discussed under Adenitis. (See Vol. II, p. 501.)

(*b*) *Tuberculosis of the Mesenteric Glands (Tabes mesenterica)*.—This is a common localization of tuberculosis in children, although, as already stated, much less so than is that in the bronchial glands. It may be combined with tuberculosis of the intestine, or less frequently occur without evident lesions there. As a rule there are no clinical manifestations apart from those of other portions of the body, especially the intestine. The symptoms will be discussed under Adenitis. (See Vol. II, p. 502.)

¹ Brit. Jour. Child. Dis., 1909, VI, 205.

(c) *Tuberculosis of the Cervical Glands*.—Tuberculosis of the external glands is exhibited in the cervical group with much the greatest frequency. It is especially common in early and later childhood, less so in infancy. The group of symptoms often associated with and frequently preceding it has for many years been described under the title "Scrofulosis," and the term is still employed in many text-books. The adenitis is usually unaccompanied by discoverable evidences of tuberculosis of the bones, lungs, or other viscera. The portal of entry is probably the tonsils, adenoid growths and chronic lesions of the pharynx (see p. 543); or there may occur much less often an extension of infection from the bronchial glands. In 106 cases in which the tonsils were removed for tuberculosis of the cervical glands, Mitchell¹ records infection of the tonsils in 41. Deterioration of the general health or attacks of acute infectious diseases, especially measles and pertussis, act as predisposing causes. The bacilli may be either human or bovine in type. (See p. 542.) The process may extend to the axillary and sometimes to other external glands. The bronchial lymph-nodes have been found involved in most of the cases coming to autopsy. Apart from the glandular affection, symptoms called "scrofulous" are described. The subject will be further discussed under Diseases of the Lymphatic Glands. (See Vol. II, p. 498.) Here only may be said that since it is certain that the involvement of the glands in "scrofula" is invariably tuberculous, and very possible that the other local conditions are in reality tuberculous lesions also, the retaining of the word in the nomenclature of diseases appears to be unnecessary and a source of confusion.

(d) *General Tuberculous Adenitis*.—This condition without other clinical manifestations is of unusual occurrence at any period of life. In it one set of glands after the other may be attacked without other discoverable lesions. The disorder is to be distinguished from Hodgkin's disease. It has been especially studied by Lesage and Pascal.²

3. Tuberculosis of the Alimentary Tract.—The disease, as stated, not infrequently attacks the tonsils and adenoid tissue and spreads thence to the cervical glands. White³ estimates the presence of tuberculosis in 5 per cent. of adenoids. The stomach is but rarely the seat of tuberculosis. The most frequent localization of alimentary infection in children is in the intestine, where the disease assumes the form of ileocolitis. (See Intestinal Ulceration, p. 798.). This is not common in early life after the age of infancy. In the majority of cases it is secondary to involvement of the lungs, and is usually associated with tuberculosis of the mesenteric glands. The small intestine is the region oftenest involved, the colon is sometimes secondarily so, and occasionally the cecum is the only part attacked. The diagnosis from other forms of ileocolitis depends chiefly upon the discovery of the tubercle bacilli in the stools or of tuberculosis elsewhere in the body.

4. Tuberculosis of the Genito-urinary Tract.—While miliary tuberculosis of the kidney is frequent in cases of general tuberculosis, clinical manifestations of tuberculous renal disease are rare in children. Tuberculosis of the testis is occasionally seen and more rarely of the female genitals. That of the penis has followed ritual circumcision. Of this occurrence Reuben⁴ has collected 42 reported instances.

¹ Jour. Pathol. and Bacteriol., 1917, XXI, 248.

² Arch. gén. de méd., 1893, CLXXI, 1, 270.

³ Amer. Journ. med. Sci., 1907, CXXIV, 228.

⁴ Arch. of Pediat., 1917, XXXIV, 186.

5. Tuberculosis of the Nervous System.—Apart from involvement of the meninges, the nervous system is not often attacked. Large, solitary tubercles are sometimes found in the brain or spinal cord. These may become cheesy or even calcareous, and may give rise to localizing symptoms, as in the case of tumors of other nature.

6. Tuberculosis of Serous Membranes.—This may occur widespread as one of the manifestations of general tuberculosis, or may predominate in, or be confined to, certain of the serous membranes, especially the meninges, the peritoneum, and the pleura. Meningitis is very common and a frequent cause of death. It is nearly invariably secondary to tuberculosis elsewhere, although often without symptoms except those of meningitis. It is most frequent in later infancy and early childhood. The clinical manifestations will be described under Meningitis (Vol. II, p. 326). In infancy involvement of the peritoneum is usually only one of the manifestations of a more general tuberculosis. After this period tuberculosis may be limited to the peritoneum, or the involvement of this be at least the chief cause of the symptoms observed. It may be apparently primary, the lesion at the portal of entry not being discovered; or much oftener secondary to tuberculosis elsewhere, as in the mesenteric glands or, less often, the lungs or other regions. (See Peritonitis, p. 852.) Tuberculous pleurisy with serous or purulent exudate is seen in later childhood; less often before this. Without effusion it is of common occurrence as one of the symptoms of general tuberculosis, or as an attendant upon tuberculosis of the lungs.

Tuberculosis of the bones and joints and of the skin will be discussed under the disorders of those regions.

Prognosis.—Tuberculosis is frequently stated to be the cause of about $\frac{1}{4}$ of all deaths. The census of the United States published in 1900 (Wilbur)¹ gives tuberculosis as the cause of death in 11.22 per cent. of the total mortality, and unquestionably the actual proportion was decidedly larger than this. On the other hand, a large number of cases which show tuberculosis at autopsy have died of other affections. The general mortality from tuberculosis has without doubt diminished in recent years. This is not so much because the patients recover more readily, although this has certainly resulted from improved methods of treatment, as because preventive measures have diminished the number of cases developing. It is noteworthy, however, as demonstrated by Behla² for Prussia, that the diminution in the number of deaths from tuberculosis in children has not kept pace with that for later periods of life; and the same was shown by Hoffman³ to be true to some extent for the Registration Area of the United States.

The prognosis of tuberculosis in children varies with the age and with the form of the disease. In the 1st year there appears to be almost no power of resistance, and the result is nearly always fatal; but in proportion as age advances this power becomes greater and arrest of the process, permanent or temporary, often takes place. Tuberculous bronchopneumonia is practically always fatal, either in the first attack or in the relapses which are likely to occur. Only isolated cases of recovery from tuberculous meningitis have been reported, and these may have been but temporary arrests of the disease. (See Tuberculous Meningitis, Vol. II, p. 334.) On the other hand, tuberculous peritonitis frequently terminates favorably, with or without operation; osseous tuber-

¹ New York Med. Journ., 1908, LXXXVIII, 798.

² Berl. klin. Woch., 1913, L, 1951.

³ Journ. of the Outdoor Life, 1913, X, 361.

culosis is often cured; that involving the bronchial lymphatic glands is in itself seldom a cause of death, and tuberculosis of the cervical glands is usually not a condition which menaces life. The prognostic indications of the tuberculin-reaction will be referred to later (p. 561).

Diagnosis.—Apart from the manifestations pertaining to, and the characteristics of special localizations of, tuberculosis, there are certain features which are suggestive from a diagnostic point of view. Careful attention should be given to the history of tuberculosis in the family as bearing upon the possibility of the inheritance of a predisposition to the disease, or still more of peculiar opportunity for the acquiring of the affection by association with tuberculous relatives. The existence of unfavorable hygienic conditions is to be sought, such as render the subjects more susceptible or occasion unusually free contact with the germs. The earlier occurrence of other diseases which may predispose is also to be considered. Especially, in the case of pulmonary tuberculosis, are to be suspected previous attacks of pertussis, measles, pneumonia, bronchitis, and grippe. A generally defective state of health of the child, and the existence of the physique which is known to be associated with tuberculosis, are also of moment.

In the case of active tuberculosis of an obscure nature, the occurrence of fever is important. In the large majority of cases some elevation of temperature is present at times. It is generally of an irregular type and often only slight; and is, of course, not conclusive, since so many other conditions are capable of producing it, especially in early life. In spite of this fact, the continual repetition of frequent rises of temperature, other causes having been sought for and excluded as far as possible, is an extremely suspicious circumstance. It is a matter of common occurrence, as I know from my own experience, to consider a general tuberculosis to be typhoid fever, until some distinct localizing process finally develops; and I recall one instance in which two eminent physicians had respectively made the diagnosis, the one of endocarditis and the other of chronic intestinal indigestion and toxemia, and which finally eventuated in a tuberculous peritonitis. Many analogous cases could easily be detailed.

A persistent increasing impairment of the general health also arouses suspicion, although not necessarily present in many forms of tuberculosis; nor of certain indication, since it may depend upon so many other factors. Chronic cough is of diagnostic value to a limited extent. It may depend upon many different causes, but tuberculosis of the lungs or pleura is always to be thought of. When paroxysmal and stenotic in character and associated with dyspnea, it may indicate tuberculosis of the tracheo-bronchial glands. Hemoptysis is so uncommon in early life that it need scarcely be considered as a possible diagnostic sign. Pleurisy with a serous effusion is always suggestive of tuberculosis when occurring in early life. The discovery of nodular masses in the abdominal cavity points, in the case of children, more to tuberculosis than to any other condition, and ascites is very probably tuberculous, if cardiac and renal diseases can be excluded.

The examination of the blood is of little diagnostic value. Slight increase in the number of leucocytes is sometimes present, the degree varying with the form of the disease. The only exception seems to be tuberculous meningitis, in which there is not infrequently a decided leucocytosis. In acute pulmonary cases the absence of a high polymorphonuclear leucocytosis tends to exclude pneumonia of other than tuberculous origin.

The x-ray examination is of very great value in some forms of tuberculosis, notably when it affects the bones and joints, tracheo-bronchial glands, lungs, and pleura. It must be admitted, however, that the pictures are sometimes misleading, and that the results of the examination must be regarded as confirmatory and the diagnosis must be in accord with other physical signs and symptoms. The existence of complement-fixation in tuberculosis is not sufficiently certain to be depended upon, and the same is true of the agglutinative reaction often present.

When the disease is of a nature which permits of the discovery of tubercle bacilli, the diagnosis is, of course, made certain. It is important, however, to be absolutely sure that the bacilli really are those of tuberculosis. They may be found in the large majority of instances of tuberculous meningitis and in many cases of tuberculosis of the lungs. Inasmuch as young children do not expectorate, special measures must be employed to obtain the sputum. For this purpose a swab of absorbent cotton or of muslin, firmly attached to a curved applicator, may be applied to the region of the glottis, and the sputum which the consequent coughing brings up can be caught upon this. Bacilli may also not infrequently be found in the stools, either in cases of intestinal tuberculosis or when the sputum has been swallowed in pulmonary disease.

The Tuberculin Reaction.—This may be obtained in various ways: (1) Tuberculin may be injected subcutaneously in a dosage of not over 0.1 milligram (.015 grain) and the occurrence of a constitutional reaction with rise of temperature noted. The procedure is valueless when fever is already present, and is not devoid of the danger of rendering active a condition which is latent. (2) The intracutaneous test is obtained by injecting 0.1 c.c. (1.6 minim) of a 1:100 solution of old-tuberculin into the superficial layers of the skin. The resulting appearances in positive cases are very similar to those seen in the cutaneous test. (3) The cutaneous reaction of v. Pirquet¹ presently to be described. (4) The percutaneous test, devised by Moro,² consists in vigorous rubbing, lasting 1 minute, into the sound skin of a 50 per cent. mixture of tuberculin and lanolin. In 24 to 48 hours red papules appear varying in number with the intensity of the reaction. (5) The ophthlmo-reaction of Wolff-Eisner³ and Calmette⁴ is obtained by dropping into the eye 1 drop (0.062) of a 0.5 per cent. aqueous solution of old tuberculin. The reaction varies from a slight reddening up to decided conjunctivitis. Owing to the damage to the eye sometimes produced, the test has with reason lost favor.

The greater convenience and equal effectiveness of the cutaneous reaction makes it easily the choice of methods. It is made by washing the skin of the upper arm with ether or alcohol, applying a drop of undiluted old-tuberculin, and then scarifying slightly through this with a sterilized needle or the special drill-shaped scarificator made for the purpose. A control scarification should be made a couple of inches distant for the purpose of comparison. A positive reaction develops within 24 hours, sometimes longer, and consists of a red, slightly indurated maculo-papule 5 mm. (0.2 inch) or more in diameter (Fig. 182). It reaches its height usually upon the 2d day and fades slowly, often leaving slight scaliness. A papule of smaller size than 5 mm. is not to be considered certainly positive (von Pirquet).⁵ If the reaction obtained is doubtful or negative,

¹ Wien. med. Woch., 1907, LVII, 1370.

² Wien. klin. Woch., 1907, XX, 933.

³ Berl. klin. Woch., 1907, XLIV, 7.

⁴ Comp. rend. acad. sci., 1907, CXLIV, 1324.

⁵ Feer's Lehrb. der Kinderh., 1914, 677.

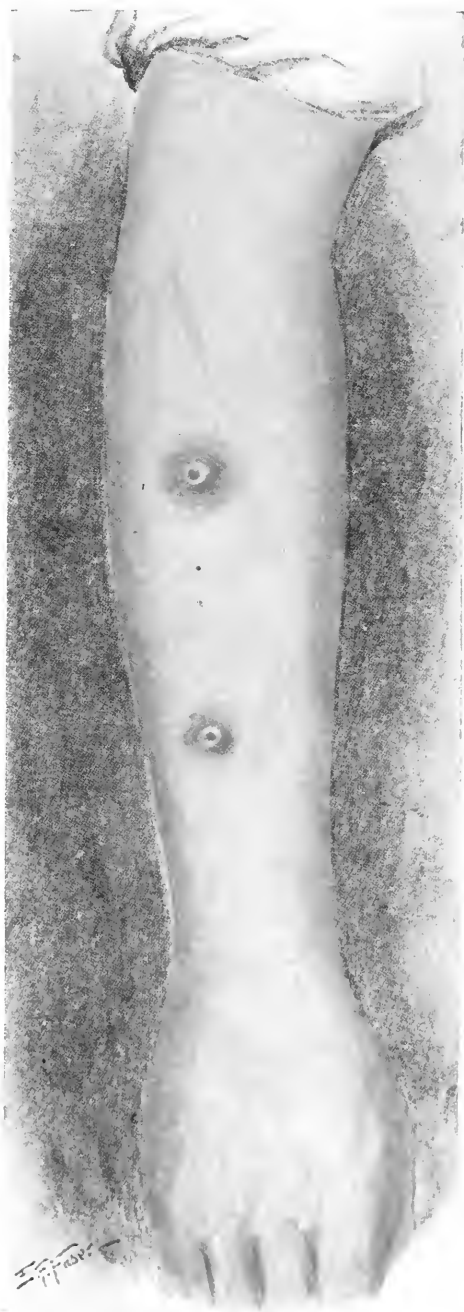


FIG 182.—THE VON PIRQUET CUTANEOUS TUBERCULIN REACTION.

Child with incipient pulmonary tuberculosis; a + reaction. The control scarification is barely to be seen, and is midway between the tuberculin reactions. (*Kolmer, A Practical Text-book of Infection, Immunity and Specific Therapy, 1917.*)

a second trial may be made in a few days or, still better, the intracutaneous test may be used.

Value of the Tuberculin Reaction.—Under ordinary circumstances it is fair to assume that a negative tuberculin reaction, especially if it is still so on the second trial, excludes the presence of tuberculous infection in the patient. In patients gravely ill in an advanced stage of the disease, the organism may be so unable to produce antibodies that no reaction takes place. Cases of general miliary tuberculosis and of tuberculous meningitis may also fail to respond to the test. There is often, too, observed an inhibitory action of some of the acute infectious diseases, notably measles, the tuberculin reaction failing to develop during their course, although positive before or after this.

A positive reaction indicates that the patient has or has had tuberculosis, and that antibodies exist in the blood. The lesion may be small, old, healed, and entirely unimportant, and this should always be borne in mind in forming a diagnosis of the nature of the symptoms present. Its lack of value for diagnostic and prognostic purposes increases in proportion as the patient is older. In infancy the finding of a positive tubercular reaction generally indicates an active process, and that very probably the symptoms observed are closely connected with this. This is especially true in the 1st year of life. It is then of serious import, although not a certain proof that the disease is advancing and that the issue will be fatal. The great frequency of the positive reaction in older children, to which reference has already been made (p. 539), shows how guarded one must be in attributing too much importance to it, so far as association with the existing symptoms is concerned.

In this connection it is interesting to consider the susceptibility to the tuberculin-reaction of those who exhibit the human or the bovine type of bacillus respectively. Although a small percentage of individuals will react with the tuberculin of only one of the two types of the bacillus, the large majority of those who give a positive reaction with one type will do so with the other also. (See contributions by Ramsey,¹ Cattaneo,² and DeLange.³)

Treatment. Prophylaxis.—The great need is that the infant be guarded against every possible contact with tuberculous subjects, even though these be the parents. No nurse with tuberculosis, healed or otherwise, should be employed. Dwellings should be thoroughly disinfected if the slightest suspicion exists of the presence of bacilli in them. The ingestion of milk from cows with tuberculosis is to be avoided, and when it cannot be made certain by veterinary inspection that the milk is above suspicion, it should invariably be pasteurized. The putting of toys and the like into the mouth of the infant must be prevented as far as possible, and the greatest cleanliness exercised in the care of its fingernails, and of the spoons, cups, rubber nipples, and other articles used in the giving of food. Promiscuous kissing of the baby by visitors is unwise. Association of an infant in its 1st year with an individual suffering from cough may be dangerous. In fact, during this period the baby is better from every point of view if kept from such contact with other individuals. When for any reason, as among the poor, it is impossible entirely to prevent contact of the infant with members of the household suffering from pulmonary tuberculosis, at least the greatest care in the collection and

¹ Amer. Jour. Dis. Child., 1915, X, 201.

² Zeit. f. Kinderh., Orig., 1913, VI, 506.

³ Nederl. Tijdschr. v. Geneesk., 1914, LVIII, 2, 438.

destruction of the sputum can be urged. The resisting power of the child must be maintained by abundant, suitable nourishment, life in the open air, plenty of sunshine, gymnastic exercises, cool bathing, suitable clothing and other hygienic measures. In this line is a temporary sojourn at the seashore or mountains if signs of debility become manifest. There is also great need in the case of delicate children for the prompt treatment of bronchitis, the removal of hypertrophied tonsillar tissue, and the avoidance of contact with pertussis, grippe and measles. In the production of immunity by tuberculin nothing of real value has as yet been accomplished. The employment of vaccines may perhaps later become of service both as a preventive and as a curative measure.

Treatment of the Attack.—This depends so greatly upon the localization and on other factors, that it can be considered, for the most part, only in the separate sections treating of the various forms of the disease. During the acute febrile stage the patient should be much of the time at rest, but this should be, if possible, in the open air. Here may be mentioned also the benefit in all forms of tuberculosis to be derived from abundant nourishment, especially milk in some form, and from change of climate, particularly to elevated, dry regions, although the seacoast is of value in many cases. Treatment in sanatoria for children, situated in a favorable climate, is often efficacious. Even where change of climate cannot be obtained, efforts can be made to ensure a life largely in the open air, as by the making use in chronic cases of open-air schools, and the like, or of similar methods employed at home (Knopf),¹ (Michael).² The employment of tuberculin of various sorts as a curative agent, of which so much was once hoped, has not obtained any settled place in the treatment of the disease, especially in children. It is but fair to state that it is highly recommended by physicians of experience; as by Wittich,³ Schlossmann,⁴ Beck⁵ and others. There would appear to be always danger of increasing the activity of the process, or of awakening it if it has been dormant. The initial dose recommended should be very small; not over 0.001 milligram (0.00015 grain). Among drugs probably the most useful is cod-liver oil, alone or combined with iodide of iron, creosote, or sometimes arsenic. If it is not well tolerated and diminishes the appetite, it should be abandoned.

CHAPTER XX

SYPHILIS

This affection, the origin of which is unknown, but which in all probability existed in ancient times, became pandemic in Europe toward the end of the 15th century. In early life it is represented by two forms, (A) Acquired Syphilis; (B) Hereditary Syphilis.

Frequency.—The influence of the disease is very great. It is true that Fruhinsholz⁶ recorded it in but 186 (1.07 per cent.) of 17,282 children under the age of 12 years, only 12 of these being proven to be acquired syphilis; and Still⁷ found hereditary syphilis in but 29 (0.6 per cent.)

¹ Med. Rec., 1913, LXXXIV, 875.

² Review in Amer. Journ. Dis. Child., 1916, XI, 162.

³ Jahrb. f. Kinderh., 1912, LXXV, 166.

⁴ Deut. med. Woch., 1909, XXXV, 289.

⁵ Zeit. f. Kinderh., Orig., 1913, VI, 439.

⁶ Rev. d'hyg. et de méd. inf., 1903, II, 1.

⁷ Pediatrics, 1904, XVI, 577.

of 4830 children under 10 years of age. The statistics of Neumann and Oberwarth¹ on 69,221 children and of Cassel² upon 17,448 infants give a percentage very close to that of Fruhinsholz. If the diagnosis based solely upon the existence of a positive Wassermann reaction be included with those determined by clinical examination, the proportion of instances of hereditary syphilis is increased decidedly, ranging from 2 to 14 per cent. according to the various statistics collected by Churchill and Austin;³ or, in the United States, from 2 to 6 per cent. Yet these figures by no means represent the actual number of cases, since they take no account of the numerous syphilitic infants who are born dead, prematurely or at term, or who die soon after birth, to say nothing of the very large number of abortions due to this disease.

Germ.—Of both forms the cause is the same: evidently a germ of some sort, since the disease is eminently infectious. Various germs have been described, prominent among them being the bacillus of Lustgarten,⁴ and the *cytotryctes luis* described by Siegel.⁵ The researches of Schaudinn and Hoffmann⁶ confirmed by many others, showed that the cause is a spirillum, the *spirochæte pallida*, or *treponema pallidum*, which is found in the primary lesions and mucous patches, and in hereditary cases in the various tissues and secretions of the body also, especially the liver.

The etiology in general, the pathological anatomy, and the symptoms of the two varieties of the disease will be considered separately.

(A) ACQUIRED SYPHILIS

Etiology.—This form is much more uncommon than the hereditary variety in early life, and yet not infrequently encountered. Infection takes place as in adults, except that it is usually by other paths than the genital tract. Kissing by a syphilitic mother or wet-nurse; nursing from a breast which is the seat of a syphilitic lesion; contact with infected clothing, drinking cups, nursing bottles, and the like, are the most frequent methods. Whether the disease can be transmitted by the milk itself of a syphilitic wet-nurse is doubtful. It has been communicated by vaccination in the days when human virus was employed, and from the mouth or instruments of a syphilitic operator performing ritual circumcision. Infection from the genitals of the mother during birth is possible but infrequent. Profeta's law—that an apparently healthy child will not acquire syphilis through kisses given by its syphilitic mother, or through nursing from or other contact with her—indicates only that the infant has acquired a certain degree of apparent immunity, because it is, in fact, already really syphilitic.

Pathological Anatomy and Symptoms.—These do not differ materially from those characteristic of adult life, age appearing to have no influence. There is an initial lesion at the point of infection, followed in due course by the usual secondary and finally tertiary manifestations. The attack is usually milder than in adults, and less severe than hereditary syphilis; widespread cutaneous eruption is not uncommon, but is more macular than papular; and there is a marked disposition in children to the development of moist condylomata.

¹ Arch. f. Kinderh., 1905, XLII, 64.

² Arch. f. Kinderh., 1909, L, 154.

³ Amer. Jour. Dis. Child., 1916, XLI, 355.

⁴ Wien. med. Woch., 1884, XXXIV, 1389.

⁵ Münch. med. Woch., 1905, LII, 1321.

⁶ Arbeiten aus d. kais. Gesundheitsamt, 1902, XXII, 527.

(B) HEREDITARY SYPHILIS

Etiology.—Of all infectious diseases this is the most frequently inherited. It may be transmitted by either parent or by both, but in the large majority of cases the father is the original source of the disease. The mother, already syphilitic at the time of conception, may infect the fetus, the father being healthy. Much less often the mother may acquire syphilis at some period during pregnancy, and then transmit it to the fetus (post-conceptional syphilis). Whether the syphilitic father can communicate it to the fetus without infection of the mother has been disputed. It is certainly of very common occurrence, so far as any ordinary evidence of maternal syphilis is concerned either at the time of parturition or later in life; yet Colles' law,¹ that the mother of a syphilitic infant will not become infected by contact with it, even though it have lesions in the mouth, shows that she has in some way become immune; and it seems very probable that this is because she is, herself, a subject of the disease, in a modified form and without symptoms. The investigations of Knopfmacher and Lehdorf² with Wassermann's complement-fixation test, confirmed by many others, show that such mothers do, in fact, give the characteristic reaction for syphilis in a large proportion of cases. If this point of view be accepted, then there is no such thing as the infection of the ovum by spore-carrying semen. On the contrary, the virus infects the mother, the placenta becomes syphilitic, and the organisms are thus allowed to pass through it to the blood of the fetus. Transmission of the disease to the fetus is, therefore, always from the mother.

Whether or not the infant of distinctly syphilitic parents will be born syphilitic depends upon various factors. When both parents are syphilitic the offspring will almost always be infected. The stage of the disease is important. If secondary symptoms are present in either parent, transmission is almost certain. If the symptoms in the parents have been overcome by treatment before the time of conception, or if they are tertiary, transmission will probably not occur, but to this there are numerous exceptions. In general, the more recent the disease in either parent, the more certain is it likely to appear in the child and the more severe are the symptoms. As regards post-conceptional syphilis, the shorter the time between infection and parturition, the less liable is the fetus to be syphilitic. In early post-conceptional infection of the mother active treatment may prevent the transmission of syphilis to the fetus. There is no satisfactory proof of the transmission of the disease to the third generation.

Pathological Anatomy.—In cases of early abortion there are often no characteristic microscopical anatomical changes whatever found in the fetus. In 62 such cases examined by Hecker³ gross lesions were discovered in only 15; *i.e.* 24 per cent., and lesions shown only by the microscope in an equal number. On the other hand, in the fetus approaching full term, and in infants dying a few days after birth, the changes are very characteristic. In children not showing symptoms of syphilis at birth, but dying of it after a few weeks or months, if often happens that few, if any, pathological changes of the internal organs are discovered. The longer the time after birth before symptoms appear the less liable are the internal organs to be found diseased. The basis of the early

¹ Pract. Observ. on Vener. Dis. and the Use of Mercury, London, 1837.

² Monatssch. f. Kinderheilk., 1909, VIII, 34.

³ Jahrb. f. Kinderheilk., 1901, LIV, 685.

alterations is a diffuse cellular proliferation arising from the perivascular connective tissue with involvement of the vessels. With this there is an interference with the development of the organs. Only later is there a tendency to the production of isolated gummata.

The most constantly present and characteristic pathological changes in *fetal syphilis* are those of the osseous system, especially the long tubular bones and the ribs. In the fetus and in young infants the lesion is usually an osteochondritis. Acquired syphilis does not exhibit this alteration. It occurs oftenest in the epiphyses of the long bones of the legs and arms (Epiphysitis). There is a widening of the cartilaginous layer through multiplication of the cells, with hardening due to abnormal calcification and, later, a softening from cellular infiltration, exuberant granulation-tissue, and necrosis with consecutive separation of the epiphysis. Sometimes suppuration occurs as a result of a mixed infection. Dactylitis is a lesion sometimes observed, especially in early childhood. In later childhood osteochondritis is not often seen, but there is found an osteoperiostitis, particularly of the skull and of the bones of the forearms and legs. This may be diffuse and hyperplastic, or it may be represented by isolated gummata, especially on the cranium. Involvement of the joints also occurs in the form of synovitis or chondroarthritis.

Enlargement of the spleen is nearly always present in the fetus or the new born. It exhibits merely hyperplasia but nothing characteristic. At a later period is observed an enlargement dependent upon interstitial changes or, less frequently, gummata. Changes in the liver are observed in the fetus and the new born about as frequently as those of the spleen. The organ is enlarged, firm, hard and irregular, with the capsule thickened. On section it is of yellowish color, sometimes with numerous whitish, milary nodules consisting of minute gummata. There is also decided increase of the interstitial connective tissue, with consequent atrophy of many of the hepatic cells, and narrowing of the branches of the blood-vessels and small biliary passages. The process may be diffuse, or limited to certain areas. After early childhood the liver less often exhibits alterations, although gummata of considerable size are sometimes found. The alimentary canal may exhibit condylomata of the tongue, and the same lesion or chronic inflammation or ulceration of the pharynx and tonsils. Cellular infiltration and interference with parenchymatous development may occur in the stomach and intestines.

A catarrhal inflammation of the nose is very frequent, and ulcerative lesions, superficial or deep, are often observed in the later stages of the disease. Perichondritis or ulceration of the larynx is sometimes seen, and exceptionally gummata may form in the trachea or bronchi and terminate in stenosis. In the fetus and in infants dying soon after birth there is often present the so-called "white hepatization" of the lung. In this a considerable portion of the pulmonary tissue appears whitish-grey, airless, and smooth on section, due to filling of the alveoli with degenerated epithelium, thickening of their walls, and increase of the interstitial connective tissue. Changes at later periods of the disease are less common, and consist of gummatous deposits or extensive interstitial inflammation. Lesions of the heart are not uncommon, and gummata and interstitial myocarditis have been seen even in infancy. The nervous system is not frequently involved. Gummata are only rarely seen in the brain and spinal cord, and chronic meningitis, chiefly basilar, is sometimes met with; but the most frequent lesion, especially in infancy is, hydrocephalus.

The genito-urinary system exhibits chronic interstitial nephritis, occasionally even in infancy. In the fetus and new-born enlargement of the kidney and parenchymatous changes are common. Gummata of the kidney are exceptional in early life. The testicle may be enlarged as the result of interstitial inflammation, and gummata are sometimes found. Chronic suppurative otitis is a frequent result of syphilitic pharyngitis. The eyes are less often involved in early life; choroiditis and optic neuritis sometimes occurring; and iritis developing even during intrauterine life. Keratitis is common among the later lesions. Multiple enlargement of the lymphatic glands is seen, especially in the groin, axilla, neck and about the elbow. The pancreas, thymus gland, peritoneum, suprarenal bodies and thyroid gland occasionally exhibit gummata, interstitial inflammation, or other syphilitic manifestations. Small cystic formations are frequently found in the thymus gland. Lesions of the skin are described under symptoms.

Symptoms.—It is convenient to divide the symptoms of hereditary syphilis into I. The Early Manifestations; and II. Later Manifestations.

I. THE EARLY MANIFESTATIONS OF HEREDITARY SYPHILIS.—Mothers with recent syphilitic infection, contracted at or near the time of conception, very commonly abort and the accident is likely to be repeated in later pregnancies. The lesions found in the fetus, born dead, have already been described. (See Pathological Anatomy.) Other infants are still-born at term. Some are born alive but with evidences of severe infection, and are usually capable of living only a few days or weeks. They are feeble, wasted, atrophic, and exhibit all the symptoms of extreme inanition. There may be present at birth coryza and a pemphigoid eruption of the skin, especially on the soles and palms, but sometimes elsewhere. Enlargement of the liver and spleen are demonstrable. The skin about the mouth exhibits fissures, and excoriations are present on the buttocks. Still other infants, although feeble and poorly developed and with enlargement of the liver and spleen do not at first exhibit the group of symptoms characteristic of syphilis, but after a few days rapidly develop them, the atrophy and inanition being prominent features from the moment of birth. The large majority of syphilitic infants, however, appear healthy and well developed at birth and symptoms appear only after an interval (*infantile syphilis*). A general estimate by Miller¹ based upon his own observations with those of Zeissl and Kassowitz would make from 50 to 65 per cent. of the cases first show symptoms in the 1st month, chiefly in the 3d and 4th weeks; from 20 to 30 per cent. in the 2d month, and nearly all of the remaining 10 to 15 per cent. in the 3d month. Generally the danger of syphilis appearing is over after 3 months but to this there are exceptions, and the first symptoms may occasionally not develop until the 4th month or even later.

The earliest symptom in the child apparently healthy at birth is usually a persistent coryza, producing the "snuffles" often described. This is soon followed by a hoarse, high-pitched and very persistent crying especially at night. In a short time cutaneous manifestations appear, often accompanied by slight fever; and then, or earlier, evidences of osseous lesions with consequent apparently paralytic conditions. Sometimes the symptoms mentioned are little, if at all, marked, and severe visceral affections are more prominent and often lead to a fatal ending. As a rule, however, decided visceral symptoms are characteristic of

¹ Jahrb. f. Kinderheilk., 1888, XXVII, 362.

fetal syphilis, and the earlier they manifest themselves in the new born the worse the general symptoms are liable to be. The majority of infants who are in good condition at birth and exhibit no syphilitic manifestations of any sort for some weeks, will present no visceral symptoms of any moment.

The promptness of development and the severity of the symptoms are, in general, proportionate to the intensity of the infection. In severe cases increasing debility and wasting begin early and advance as the disease progresses, and infants may finally exhibit all the evidences of extreme marasmus. In milder cases the general health and nutrition may remain unaffected.

The symptoms referred to, as well as others which develop, require more detailed description:—

Cutaneous Symptoms.—The severest symptom of this nature, which has been designated pemphigus syphiliticus neonatorum, is a bullous



FIG. 183.—HEREDITARY SYPHILIS, BULLOUS ERUPTION ON SOLES OF FEET.

Courtesy of Dr. J. F. Schamberg.

eruption, with bloody or purulent contents, oftenest found on the palms and soles, but at times widespread and causing extensive exfoliation of the skin (Fig. 183). Most instances show the lesion at birth and the cases generally terminate fatally. In milder cases of syphilis there is no cutaneous eruption at first and the earliest evidence of it in any form appears generally about a week after the development of the coryza. The eruption may be circumscribed or diffuse. The *circumscribed* form is much the same as that found in acquired syphilis. It is oftenest maculo-papular, and occurs in the shape of small, slightly elevated, pea-sized or larger macules associated with papules more or less numerous, situated especially upon the lower extremities, the face, scalp, neck, and flexor surfaces of the upper extremities, the palms and soles. Sealing may be present upon the surface. It is unattended by itching and varies in color from coppery-red to brown or yellowish according to the age of the lesion. Absorption may take place in the center, producing in some cases a well-marked annular appearance (Fig. 184). In certain regions there may develop flattened elevations with moist surfaces—the “moist condyloma” or mucous patch. These are commonest about



FIG. 184.—HEREDITARY SYPHILIS, MACULO-PAPULAR ERUPTION.
Unusual degree of annular appearance. *Courtesy of Dr. J. F. Schamberg.*



FIG. 185.—PAPULO-PUSTULAR SYPHILODERM.
Courtesy of Dr. M. B. Hartzell.

the mouth, the anal region and the genitals, but may develop in any situation where the skin is thin and delicate, such as behind the ears, between the fingers and about the navel. The condyloma, however, is not the earliest eruption of hereditary syphilis, but is particularly characteristic of relapses. A more papular eruption of a brown-red color is seen oftenest upon the palms and soles and the forehead. Less often seen, and in severer cases, is a papulo-pustular eruption (Fig. 185) which may terminate in ulcerated or ecthymatous lesions. The roseola of acquired syphilis is rarely if ever witnessed in the hereditary form.

The *diffuse* eruption is common and very characteristic, and is not observed in acquired syphilis. It consists of a diffuse infiltration of the



FIG. 186.—SYPHILITIC SCALING OF FEET.

From an infant aged 6 weeks, in the Children's Medical Ward of the Hospital of the University of Pennsylvania. At the age of 2 weeks snuffles and cutaneous eruption developed. The latter seen as macules on the face and limbs and as a red diffuse infiltration on the soles of the feet. The illustration shows the typical sealy appearance following.

skin, situated usually on the palms and soles, face, scalp, genital and anal regions, and the flexor surfaces of the thighs. It presents a somewhat shining surface of a copper-red or brownish-yellow color and renders the skin distinctly stiffened. Slight desquamation is often present. In other cases it produces extensive desquamation in small scales or larger flakes (Fig. 186). Sometimes when inflamed it suggests the appearance of eczema, with formation of crusts but differs in the presence of stiffness of the skin and the absence of the intensely red color of the latter disease.

Fissures in the skin are very characteristic early symptoms. They occur especially about the mouth and eyelids at, the alae of the nose, and at the anus. Those about the mouth are situated on the lips, and extend a short distance into the skin beyond. They are often arranged radially, especially at the labial angle (Fig. 187), are narrow, infiltrated, rather deep, painful, bleed readily and are often covered with crusts. When healed they may leave very characteristic linear scarring.

The nails are often dry, shriveled, narrowed and curved in a sharp

transverse curve, exhibit transverse fissures, and may fall off. In other cases inflammation about the nail occurs (syphilitic paronychia). There



FIG. 187.—FISSURES ABOUT THE MOUTH IN HEREDITARY SYPHILIS.

Female child, aged 6 months, in the Children's Ward of the Hospital of the University of Pennsylvania. Previous history not discoverable, except that the child had been ill since its 1st month. Under treatment for a month with mercury and arsphenamine; no improvement; death.



FIG. 188.—SYPHILITIC ALOPECIA.

Courtesy of Dr. J. F. Schamberg.

may be loss of hair from the scalp (Fig. 188), eyelids and eyebrows. This is present at birth in fetal cases.

Mucous Membranes.—Lesions of the mucous membranes, apart from the rhinitis to be described, are not frequent early symptoms in infancy and are usually not seen until the infant is some months old. Broad condylomata may develop on the lining of the mouth, the tongue and the vulva, but oftener, as stated, upon the skin near the mucocutaneous border. Ulcers, general superficial, may occur upon the mucous membranes of the mouth, pharynx, and elsewhere. "Snuffling" is generally the first manifestation of the disease, and a very persistent one. It is nearly always present, and often at birth, and may be the only symptom. The nasal mucous membrane is swollen, and respiration may be much interfered with, rendering sucking difficult and sleep disturbed. Later there develops a serous, mucous or muco-purulent discharge, often tinged with blood, and thick crusts block the nostrils and interfere still further with breathing. The mouth may be dry from the constant mouth-breathing. Hoarseness may attend the coryza, being sometimes one of the earliest symptoms. Hemorrhages occasionally occur from the mucous membrane and from some of the cutaneous lesions, or sometimes in the internal organs. Some of the cases of hemorrhagic disease in the new born very probably owe their origin to syphilis.

The **lymphatic glands** may show moderate enlargement, especially in the axilla, elbow, groins and neck. This enlargement of the cubital glands is very common and suggestive, but not proof of the existence of syphilis. Syphilitic glands appear as multiple, hard bodies, without tendency to suppurate. From a study of 390 children, Reicheneker,¹ concludes that epitrochlear adenitis is most frequently due to syphilis, and Götzky² believed that in infancy it is almost certainly dependent upon this disease. It should, however, be stated that these views do not meet universal acceptance, and the frequency of enlargement of the lymphatic glands in congenital syphilis is probably over-estimated. The *teeth* are often late in their eruption and decay early, but show nothing characteristic.

The **osseous system** (see also Vol. II, p. 450, Syphilis of the Bones) is frequently affected in early life, the most characteristic symptom being an osteochondritis. This lesion really begins in fetal life and lasts a variable time, although the clinical evidences of it are usually not discoverable at birth. It appears first as a tender swelling at the junction of the shaft and epiphysis, oftenest in the extremities, and may advance to complete separation of the epiphysis with temporary loss of power and pain on passive movement, suggesting paralysis;—the syphilitic pseudo-paralysis described by Parrot.³ This paralytic condition occurs oftenest in the earlier months of life, and usually in one arm, which hangs motionless with the forearm in full pronation and the palm of the hand turned outward. The inability to move the limb depends upon pain, not upon loss of power. The process may involve the joint itself with a secondary suppuration (Fig. 189), or a large portion of the shaft of the bone may be attacked by the disease. It is undetermined whether all the cases of pseudo-paralysis depend in reality upon an osseous lesion. A form of osteitis of the bones of the fingers (dactylitis), and, less often, of the toes, has been described by Hochsinger⁴ as occurring during the early symp-

¹ Le nourrisson, 1915, III, 193. Ref. Brit. Jour. Child. Dis., 1916, XIII, 60.

² Zeit. f. Kinderh. Orig., 1913, VII, 113.

³ Arch. de phys. norm. et path., 1871-2, IV, 319.

⁴ Pfaundler and Schlossmann. Handb. d. Kinderheilk. 1906, I, 2, 916.

toms in infancy. It begins chiefly in the proximal phalanx, is bilateral, and never advances to suppuration. (See Syphilitic Dactylitis, Vol. II, p. 451, Fig. 392.) A more destructive dactylitis may develop at a later period in infancy. It is described under the later symptoms of syphilis.



FIG. 189.—SYPHILITIC EPIPHYSITIS WITH SECONDARY SUPPURATION.
Same case as Fig. 187.

Craniotabes is an occasional osseous lesion seen in infancy. It consists of thin, softened spots, particularly in the occipital region, but is not entirely pathognomonic of syphilis since it may occur in rachitis as well.



FIG. 190.—SADDLE-NOSE IN EARLY SYPHILIS.
Same case as Fig. 187.

(See p. 587; Vol. II, p. 428.) Flattening of the bridge of the nose producing the "saddle-nose" is sometimes an early symptom (Fig. 190). It may be only an apparent flattening at this stage, but often depends upon actual

involvement of the cartilages and bones. The affections of the *eye* and *ear* among the early symptoms of syphilis, already referred to in discussing pathological anatomy, consist principally of iritis, choroiditis, optic neuritis and purulent otitis.

Visceral lesions are, as indicated, early symptoms more characteristic of syphilis of the fetus and the new born than of the disease first showing itself in infants past this period. They date from intra-uterine life and are well-marked only in unfavorable cases. The commonest clinical manifestation is decided enlargement of the liver, the organ presenting a hard edge on palpation and projecting considerably farther downward than normal. Ascites and icterus occasionally result. (See Congenital Obliteration of the Bile Ducts, p. 273.) The spleen also may be much enlarged. Symptoms depending upon lesions of the nervous system are not very common early manifestations. Among them are those of meningitis and the consequent hydrocephalus. This may develop at the time of the early cutaneous eruption, or later during relapses. A functional disturbance is, however, very frequent; namely the great wakefulness with persistent crying, especially at night. This may very probably be due, at least to some extent, to the pains in the bones. Albuminuria may or may not depend upon the existence of syphilitic nephritis. According to Hintzelmann¹ evidences of nephritis were found in 14 out of 41 syphilitic infants.

The **general nutrition** suffers badly in severe cases. With the development of other symptoms there is a rapid emaciation with marked anemia, the blood showing diminution of the red cells and hemoglobin, and increase of the leucocytes. The skin often exhibits, besides the pallor, a peculiar *cafe-au-lait* color, either throughout or in certain localities, especially the face. The veins of the scalp are sometimes much dilated. The debility is great, and a fatal ending may occur dependent upon a marantic state, and entirely independent of any discoverable affection of the digestive apparatus or fault with the food. In cases which recover, the anemia persists after the disappearance of all other symptoms. Sometimes the marasmus is the earliest or even the only symptom, and not infrequently it does not yield at all to antisiphilitic treatment.

The *frequency of the occurrence* of the different early symptoms, according to the observations of Still,² shows snuffling 70 per cent.; cutaneous eruptions 69 per cent.; splenic enlargement 45 per cent., and quite decided in 22 per cent.; affections of the eye 15 per cent.; laryngitis 14 per cent.; epiphysitis 11 per cent.; orchitis 8 per cent. Hochsinger³ found enlargement of the liver in 31 per cent. of the cases, always accompanied by splenic enlargement.

II. LATER MANIFESTATIONS OF HEREDITARY SYPHILIS—The symptoms just described would be classified as secondary ones in the acquired disease, there being, of course, no primary lesion in the inherited affection. They appear for the most part in the 1st and 2d months of life, and most or all of them disappear entirely and permanently under proper treatment in a few weeks. Unless, however, treatment is long continued, recurrence occurs in a few months, or by the end of the 1st year or later, exhibiting some of the original cutaneous and other symptoms, with a special tendency, however, in early childhood to the development of condylomata. That this may take place in spite of treatment is shown by the fact that

¹ Zeit. f. Kinderh., Orig., 1913, IX, 27.

² Lancet, 1904, II, 1402.

³ Pfäundler and Schlossmann, Handb. d. Kinderheilk., 1906, I, 2, 921.

in 208 cases of congenital syphilis observed by Hochsinger¹ during a period of more than 4 years, 131 (63 per cent.) exhibited recurrence, and of these 112 (54 per cent.) had undergone treatment carried out in a thorough manner.

In addition to those described, new symptoms may appear which would be classified, for the most part, as tertiary in acquired syphilis; prominent among them being gummata in various regions. These are seen usually after the period of early childhood. In other cases the later symptoms develop about the time of puberty or even after this, without any of the earlier secondary symptoms having been observed, or, if noticed, so slight that their nature was unrecognized. To this form of the



FIG. 191.—GUMMATA OF ELBOWS AND CHEST IN LATE HEREDITARY SYPHILIS.
Courtesy of Dr. H. R. Wharton.

disease is sometimes applied the title "*Syphilis hereditaria tarda*." The existence of such a condition without previous symptoms has been much disputed, as is, indeed, the proper application of the name.

The principal later symptoms of syphilis are the affections of the permanent teeth and of the bones, eyes and ears, and various lesions of the skin, principally of a condylomatous or gummatous nature. Some of the later symptoms must be described more in detail:—

The *general nutrition* suffers, there being a decided retardation of growth, general impairment of vigor, delayed puberty, and anemia. The last may persist after other symptoms have disappeared.

Osseous changes are present, usually represented by localized gummata or by a hyperplastic osteoperiostitis. This latter affects chiefly the long bones of the extremities, and the cranium. It produces thickening, tenderness, and pain which is most marked at night. In the tibia, which

¹ Ergebnisse. inn. Med. u. Kinderh., 1910, V, 84.

is the bone oftenest involved, there is a thickening and curvature of the anterior edge, resulting in the well-known *sabre-tibia* (Vol. II, p. 452, Fig. 393). When the cranium is attacked there results a diffuse thickening with corresponding deformities (Fig. 393). A dactylitis affecting one or more fingers and often going on to necrosis, and in which the soft parts are also involved, is seen oftener in early childhood than at a later period. It attacks usually the proximal phalanx. (See Dactylitis, Vol. II, p. 451.) Gummata of the bones (Fig. 191) may develop in various regions and persist for a long time; finally breaking down and forming ulcers, or being absorbed and leaving depressions. On the skull, especially the parietal and frontal regions, they form large nodular masses. These or the osteoperiostitis referred to produce decided alterations in the shape of the head—the “keel-shaped” cranium, with central frontal deposit, and the “natiform” cranium, with bilateral parietal deposits. Hochsinger¹ considers these as more commonly early symptoms. A frequent site of gummata in child-



FIG. 192.—SADDLE-NOSE IN LATER HEREDITARY SYPHILIS.

Courtesy of Dr. Harry Lowenburg.

hood is the nasal septum and hard palate, in both of which localities breaking down is liable to occur. The loss of tissue is sometimes widespread and the palate may be perforated or much of the nose destroyed. The saddle-nose, described as an occasional early symptom, may develop in childhood also, as the result of destructive necrosis in the bone and cartilage (Fig. 192). A swelling of the knee-joints is a not uncommon distinctly later symptom. It is bilateral and may be either a hydrarthrosis (Fig. 193) or a hyperplastic synovitis, and results finally in ankylosis. Less often the ankle is attacked, but any joint in the body may be affected.

The *skin* may exhibit syphilitic tubercles and gummata in various situations, but not so frequently as in acquired syphilis. They tend to break down and leave ulcers, often covered by thick crusts and finally replaced by scars. A papular eruption of the face may develop during relapses. Condylomata are, as stated, the cutaneous manifestations particularly liable to appear during relapses, and may, indeed, be the only cutaneous lesion after the 1st year and in early childhood. They are the commonest of all symptoms at these times but are not frequent

¹ Pfaundler and Schlossmann; *Handb. d. Kinderh.*, 1906, I, 2, 920, 931.

after this period. In general, the longer the time elapsed since infancy, the less pronounced as symptoms do cutaneous eruptions become.



FIG. 193.—HYDRARTHROSIS IN LATER HEREDITARY SYPHILIS.
Courtesy of Dr. J. F. Schamberg.

The incisor teeth of the second dentition, especially the two upper central ones, show very characteristic changes, described by Hutchinson¹



FIG. 194.—HUTCHINSON'S TEETH IN LATER HEREDITARY SYPHILIS.
Courtesy of Dr. J. F. Schamberg.

(Fig. 194). They are far apart and small, with notching of the cutting surface. In other cases they are tapering and characteristically "peg-

¹ Brit. Med. Journ., 1858, II, 822.

shaped." Mucous plaques and ulcers appear on the mucous membrane of the mouth, tongue and pharynx, and alterations of the larynx may develop.

Enlargement of the lymphatic glands, referred to as seen in infancy, becomes more common after the 1st year. The *nervous system* is involved in many diverse ways in the later symptoms of hereditary syphilis, although not as frequently as in adults. The pseudo-paralysis described appears exceptionally to be of nervous origin. Paralysis of the cranial nerves may occur dependent upon meningitis, encephalitic processes the result of endarteritis, or gummatous growths. Retarded mental development perhaps not appearing until later childhood, idiocy, and epilepsy have been described, and juvenile paresis or tabes is sometimes observed.

The *eye*, likewise, exhibits late symptoms. Chronic interstitial keratitis is one of the most frequent of these. It generally occurs in later childhood, attacks both eyes, and produces slight cloudiness, or even entire opacity of the cornea, which, however, usually disappears under treatment. Choroiditis, iritis, and retinitis may occur. Hearing may be affected by chronic otitis, or, in older children, by a disturbance which is of nervous origin, perhaps labyrinthine. The latter is very characteristic. It may result in deaf-mutism. Syphilis of the internal organs may also be among the later symptoms, dependent upon interstitial changes or the formation of gummata. Decided enlargement of the liver and spleen may occur, and combined with this is alteration of the blood, great anemia developing with diminution in the number of red blood-cells and hemoglobin and increase of leucocytes; and a clinical picture may result suggesting Banti's disease (Osler).¹ Interstitial nephritis may develop, and the pancreas and testicles sometimes exhibit lesions.

Reviewing the later symptoms described it will be noticed that some are characteristic of relapses in infancy and early childhood, while others, chiefly of true late hereditary syphilis, appear oftenest about the period of puberty. Among the former are condylomata of the skin and mucous membranes, debility, retardation of growth, deformities of the skull, dactylitis, and enlargement of the lymphatic glands. Among the latter are osteoperiostitis, gummata in various regions including the viscera, affections of the teeth, keratitis, labyrinthine deafness, affections of the knee-joints, and retarded mental development and other psychic disturbances. Gummata may, however, occur at an earlier period, and visceral involvement may take place in the early relapses or as a quite late symptom, as well as being seen in the new born and the fetus. Indeed there is no sharply dividing line between the ages at which the various later symptoms may show themselves. It is rather the stage of the disease which is to be considered.

Prognosis.—The prognosis of *hereditary syphilis* varies with the severity of the infection, and with the promptness and thoroughness of treatment. A large proportion of syphilitic mothers abort, or are delivered of still-born children, and those infants born alive with decided evidences of fetal syphilis nearly always die soon. Taking all cases together the death-rate is high. Coutts² in 1102 syphilitic pregnancies reported 376 abortions and 396 early deaths, making a total death-rate of 70.05 per cent., without including the deaths occurring later in life. In a series of 414 pregnancies reported by Le Pileur³ 151 aborted, and

¹ Clin. Journ., 1914, XLIII, 462.

² Lancet, 1896, I, 971.

³ Thèse de Paris, 1851. Ref., Fournier, 312.

of 260 infants born alive 141 died in a very short time. The total mortality equalled 71.26 per cent. These two series occurred in hospital practice. Apart from this, however, the death-rate usually does not reach these figures. Thus, in a series of 1127 pregnancies in syphilitic families, reported by Fournier¹ occurring in the civic population of Paris in private practice the mortality in the offspring, including abortions, equalled but 42 per cent. The majority of infants apparently healthy at birth and showing no symptoms for some weeks, can be saved by energetic treatment. In general, the less recent the disease in the parents, the better the state of nutrition in the infant when the symptoms appear, the greater the delay in the appearance of these symptoms and the less their intensity, and especially the earlier antisymphilitic treatment is commenced after birth, or preferably before it, the greater will be the probability of the child living.

Very important, too, is the influence of a supply of suitable nourishment, especially breast-milk, children fed in this way having a far better chance of survival. It is noteworthy, also, that the later children of syphilitic parents oftener survive than do the earlier ones. Death is due not so much to the lesions themselves, unless the viscera are decidedly involved, as to the profound effect which the virus exerts upon the constitution, with the general asthenia which results and the ease with which complicating processes, such as sepsis, pneumonia, intestinal disorders, and the like, develop and determine a fatal ending. The degree of the marantic symptoms constitutes probably the most important prognostic guide. There is always, too, to be remembered the tendency to repeated relapses, these sometimes occurring in spite of thorough specific treatment. They constantly diminish in intensity, however, if treatment is persisted with.

There is a tendency for symptoms of the tertiary type to appear in later childhood, even in cases which have been apparently entirely cured earlier, although still more so in those who have never received early treatment. Most of these cases yield to treatment unless severe visceral manifestations are present, especially of the kidneys and liver. The nervous manifestations, too, are often entirely resistant.

The *duration* of the disease is very uncertain. Under proper treatment in favorable breast-fed subjects the characteristic cutaneous eruptions generally disappear in 3 to 4 weeks; the rhagades, glandular swelling and snuffling lasting a longer time, the last mentioned being one of the most resisting symptoms. The asthenia and anemia, however, may remain after other symptoms have disappeared, and the child may die of causes not directly syphilitic. Only long persistence in treatment can prevent the return of symptoms or the development of new ones. Untreated infants lose the cutaneous eruption in a few weeks but soon relapse. The later symptoms of the tertiary type yield only slowly, but the prognosis as regards life is much better, Rabdl² finding only 9 deaths in 93 cases. The chance of ultimate recovery under satisfactory continued treatment is well illustrated in the statistics of Hochsinger:³ Of 263 cases kept under observation for from 4 to 12 years 79 had died, 112 exhibited symptoms of some sort, not always syphilitic, and 72 were entirely free from symptoms of any kind.

The prognosis of *acquired syphilis* in infancy is very much better than

¹ L'hérédité syphilitique, 1891, 309.

² Lues hereditaria tarda, 1887. Ref., Gerhard's Handb. d. Kinderkr.; Syphilis, 389.

³ Ergebn. d. inn. Med. u. Kinderh., 1910, V, 125.

of the hereditary disease, owing to the absence of the severe constitutional impression made by the latter. Fournier¹ saw only 1 death in 42 cases.

Diagnosis.—The diagnosis is easy in typical cases with the group of characteristic symptoms. In the fetus and new born it rests principally upon the enlargement of the spleen and liver, coryza, and osteochondritis. In early infancy the most representative symptoms are snuffling, hoarseness, cutaneous eruptions, fissures about the mouth and anus, and the debilitated condition present from birth or developing later. Relapses are characterized by return of symptoms or the development of condylomata and sometimes of visceral involvement. Among the much later diagnostic symptoms of hereditary syphilis the most important are the alteration of the teeth; the development of gummata in various regions, especially the cranium and the tibiae; interstitial keratitis; deafness of central origin; depression of the bridge of the nose from destructive lesions; ulcerations of the interior of the nose or of the palate; enlargement of the lymphatic glands; thickening and curvature of the tibiae; decided hyperplasia of the liver and spleen, and general retarded development.

In mild cases of syphilis where no characteristic group of symptoms is present, the diagnosis apart from serum-tests is difficult. The known presence of syphilis in the parents, or the occurrence of repeated earlier abortions may confirm the importance of a few suspicious symptoms in the infant. So, too, response to antisypilitic treatment is important diagnostically.

Cutaneous symptoms may be very little marked in the syphilitic cases and, when present, are carefully to be distinguished from various non-specific affections of the skin. The maculo-papular, cutaneous eruptions of syphilis differ from those not syphilitic chiefly in their situation and their color. In syphilis they are principally found on the face and the lower extremities, and exhibit always a tendency to a coppery or raw-ham tint. The diffuse syphilitic infiltration sometimes resembles eczema intertrigo, but is distinguished by the absence of the intense red color of the latter and the presence of a coppery or yellowish-brown tint and a tendency to scaling, combined with the evidence of infiltration of the skin which produces a decided hardness and stiffness. Syphilitic pemphigus is situated on an indurated base and generally occupies the soles and the palms, thus distinguishing it from other pemphigoid eruptions in the new born.

Coryza in the early weeks or months of infancy, without fever and not yielding to ordinary forms of treatment, is suspicious, even though no other symptoms are discoverable. Infantile scurvy may be mistaken for the osteo-chondritis of syphilis. In the latter, however, the lesion is nearer the epiphysis, and the process usually begins at an earlier age.

The value of the complement-fixation reaction in the diagnosis of syphilis, as applied by Wassermann, Neisser and Bruck² and as modified by others, has been supported by the testimony of numerous later investigators. It would appear that although a positive Wassermann reaction is of very great importance a negative result is of decidedly less value. When for any reason it cannot well be applied to the infant, the test may be made upon the mother. The performing of the test requires, however, much training and skill, and even with this it is probable that it cannot be absolutely relied upon in the absence of clinical symptoms.

¹ La syphilis héréditaire tardive, 1886, 600.

² Deutsch. med. Wochenschr., 1906, XXXII, 745.

It must be looked upon as corroborative evidence. (See review by Towle.)¹ Symmers and Darlington² after a long series of observations concluded that a negative reaction was present in from 31 to 56 per cent. of cases with characteristic post-mortem lesions of syphilis, and a positive reaction in at least 30 per cent. of cases without syphilitic lesions found at autopsy. It is to be noted, too, that not infrequently the Wassermann reaction does not appear until after the first few weeks of life, even when prominent symptoms of syphilis are present; while, on the other hand, a temporary, more or less positive reaction may be obtained in various non-syphilitic affections, among them tuberculosis, scarlet fever, leprosy, cachectic conditions and certain cutaneous disorders.

The value of the Noguchi³ luetin cutaneous test is still under dispute. An analysis by Comby⁴ of publications upon the subject indicates that it has not fulfilled expectations. As there is, however, decided evidence in its favor from other sources (Wolfsohn,⁵ DeBuys and Lanford,⁶ and others), further study is required.

The differentiation of the hereditary from the acquired form depends principally upon the early occurrence of visceral lesions and the existence of epiphysitis, pseudoparalysis, fissures, coryza, pemphigus, diffuse infiltration of the skin, or other symptoms characteristic of the inherited disease but not seen in acquired syphilis. In acquired syphilis, on the other hand, there occur a primary lesion, visceral involvement later in the attack and less frequently, and roseola.

Treatment. Prophylaxis.—According to the advice given by Fournier⁷ a syphilitic adult should undergo energetic treatment more or less continuously for from 3 to 4 years after the onset of the disease, and for 2 years there should have been no symptom whatever discoverable. Marriage may now take place, although there is even then no absolute certainty that the disease will not be transmitted to the offspring. If a woman becomes pregnant and her husband is syphilitic, she should at once undergo continuous treatment throughout pregnancy, whether or not she herself exhibits symptoms. If she has formerly shown evidences of syphilis, but appears to have recovered, she should still receive treatment. It is probable that by these means the infant will escape the disease.

All healthy infants should be guarded with care against the acquiring of postnatal syphilis especially from a syphilitic wet-nurse, and a thorough examination of the nurse should be insisted upon, including an examination for the Wassermann reaction. It is probable that an apparently healthy infant, born of a syphilitic mother may be suckled by her without danger of infection (Profeta's Law). At the same time, in view of the difference of opinion still regarding this, the immunity of the infant should not be trusted absolutely, and the mother should receive treatment and should avoid caressing the infant or other unnecessary contact with it. Promiscuous kissing of the baby by friends or strangers is also to be avoided.

It has been denied that hereditary syphilis is contagious, and it seems

¹ Amer. Journ. Dis. Child., 1912, IV, 180; 1914, VII, 318.

² Journ. Amer. Med. Assoc., 1918, LXX, 279.

³ Jour. Exper. Med., 1911, XIV, 557.

⁴ Arch. de méd. des enf., 1915, XVIII, 602.

⁵ Journ. Amer. Med. Assoc., 1913, LX, 1855.

⁶ Amer. Journ., Dis. Child., 1916, XII, 387.

⁷ Syphilis et Mariage, 1880.

certain that the transmission of the disease by infants with this form of the affection is uncommon. Nevertheless it is equally certain that such transmission can at times occur and syphilis be acquired by other children of the family through kissing the affected infant, or through contact with contaminated spoons, cups, toys and the like. Werther¹ records 10 cases in which the nurses were infected by the infant.

Treatment of the Disease.—Immediately after birth in the case of infants whose parents are known to be syphilitic; or, in other cases, on the first evidence of the disease in the infant; treatment with mercury should be commenced. The drug may be administered in various ways. A popular method is that of rubbing into the skin daily 5 to 10 grains (0.32 to 0.65) of mercurial ointment, selecting different situations upon different days, as the axilla, abdomen, groins, thighs, etc., in order to avoid the production of cutaneous irritation. A favorite method is to place the ointment upon the binder, and thus keep it in contact with the abdomen. The skin should be washed with soap and warm water before every inunction, and the ointment gently rubbed in for 10 or 15 minutes. In order also to avoid irritation, if the skin is the seat of any extensive cutaneous eruption, the ointment may be replaced by the sublimate bath, using 5 to 15 grains (0.32 to 0.97) of the bichloride of mercury to each bath of 5 gallons (19 liters) given daily. The water should be heated to from 100° to 105°F. (37.8° to 40.6°C.) and the child should remain in it for from 5 to 15 minutes.

Instead of these methods, or in addition to them, mercury may be given internally, either as calomel, $\frac{1}{10}$ of a grain (0.0065); the protiodide $\frac{1}{20}$ to $\frac{1}{10}$ of a grain (0.0033 to 0.0065); hydrargyrum cum creta, 1 to 2 grains (0.065 to 0.13) or corrosive sublimate, $\frac{1}{100}$ to $\frac{1}{50}$ of a grain, (0.0006 to 0.0013) 3 or 4 times a day. If the mercury produces diarrhea, its administration should be stopped for a few days or a very small amount of opium given. Corrosive sublimate is rather more prone than the other preparations to occasion digestive disturbance. Salivation rarely occurs in early infancy, and less frequently in children than in adults.

Mercurial treatment to be effective and lasting must be persisted with for 1 to 2 years, if it does not produce stomatitis or anemia and debility. If any such effects develop it should be stopped for a time. In any event it is often better to allow brief periods of intermission to occur, and it is not necessary to employ the treatment so energetically during the whole of the period. Treatment should be given at intervals during the succeeding years of infancy and early childhood, and again at puberty, in order to insure a continued absence of symptoms.

Arsenic, especially in the form of arsphenamine in some of its forms (salvarsan, arsenobenzol, etc.), has come into great prominence in the treatment of syphilis. Of its power there is no question; but the preponderance of opinion appears to be that for hereditary syphilis it is not as valuable as is mercury, although its action is often more rapid; and that success is obtained only when the administration of mercury is combined with it. Its employment is also not without danger in nurslings, and a number of deaths have been reported. Later in childhood, and in severe cases in infancy when a rapid effect is urgent, it often fills a useful place. It is irritating to the tissues, and should be given intravenously in doses of 0.02 to 0.05 grams (0.31 to 0.77 grains) for very young infants, and in proportionately larger amounts for those older. There is, however, a wide variation in opinion regarding the size of the dose. Treatment

¹ Münch. med. Woch., 1918, LXV, 71.

with mercury should be combined with the arsenical preparation; giving the latter every 14 days or oftener, the opinion regarding this being at variance, and the mercurial persistently. From 4 to 6 arsenical injections may be needed, the time for the cessation of the treatment being determined, as a rule, by the disappearance of the Wassermann reaction. In this connection is to be observed, however, that this disappearance is often very difficult to accomplish, especially in late hereditary syphilis.

Iodide of potassium or of sodium is of service only for the distinctly later manifestations of syphilis, such as occur in later relapses; and at any period when the viscera or the bones are involved. The earlier relapses, with condylomata, require the use of mercury. The employment of the iodides depends upon the nature of the symptoms, irrespective of whether they occur in infancy or after it. They may be given to infants in doses of from 2 to 10 grains (0.13 to 0.65) 3 times daily; alone or, often better, combined with the external use of mercury. The treatment must be persisted with for a long time. The combination with mercury is especially indicated in visceral syphilis.

Very important at all periods is treatment of a general tonic nature. In early infancy the general nutrition must be supported or increased by suitable nourishment, especially by the use of human milk, and by the use of all hygienic measures calculated to increase the vigor, which so often falls below normal in syphilitic infants. For the anemia and debility which commonly develop among the later symptoms, iron and cod-liver oil are of value, and may with benefit replace for a time the administration of mercury. For such cases, too, exposure to fresh air and sunshine and the employment of similar hygienic measures, with abundant nourishment, are especially important.

Local Treatment.—Many of the lesions require local measures to hasten their cure. Condylomata and cutaneous ulcerations and fissures may be dusted with calomel, covered with mercurial ointment, or painted with a solution of nitrate of silver. Ulcers on the mucous membranes require careful cleansing followed by the application of calomel or, occasionally, nitrate of silver. Gummatous ulcerations are well treated with iodoform or mercurial applications. The coryza is often benefited by removing crusts from the nose, followed by the application of a diluted yellow oxide of mercury ointment or by the insufflation of calomel well diluted with sugar (1:20).

SECTION III

GENERAL AND NUTRITIONAL DISEASES

Here may be classified a number of disorders, the cause and nature of which is not clearly understood, or which seem to belong properly to no other category. Of these, rheumatism is now very frequently placed among the acute infectious disorders; yet, inasmuch as its position here is not yet positively proven, and on account of its relationship to certain other affections, it has been thought best to place it provisionally in the class now to be considered. Rachitis, scorbutus and some forms of malnutrition and infantile atrophy are best classified as nutritional disorders, and may be placed here; as may also be diabetes, the various diatheses, acidosis and, provisionally, pellagra. Obesity, which could properly be considered in this connection, will be discussed under Diseases of the Internal Secretions.

CHAPTER I

RACHITIS

(Rickets)

Rickets, undoubtedly of earlier existence, was first named Rachitis and clearly described by Glisson.¹ It is very widely spread in civilized countries, although its prevalence varies greatly with the locality. A large proportion of infants brought to the out-patient departments of hospitals in many of the larger cities of Europe and America exhibit the disease. Morse² found it in 79.5 per cent. of 400 infants in Boston, and Schmorl³ in Dresden evidences of the disease at autopsy in 89.4 per cent. of 345 children from 2 months to 4 years of age. Practically all (96.6 per cent.) of those from 4 to 18 months exhibited the disease. It was estimated to be present in 90 per cent. of the infants in Hamburg (Volland);⁴ 74.9 per cent. of those of the poorer classes in Dundee (Foggie);⁵ 86 per cent. of infants in Riga (Mey),⁶ but in only 13.5 per cent. in Christiania (Quisling).⁷ It is essentially a chronic disease of nutrition, affecting the bones most strikingly; but general in its nature and involving as well the ligaments, muscles, mucous membranes, nervous system and other parts of the body.

Etiology.—This is not known with any certainty, in spite of the great amount of investigation upon the subject. Many different factors appear to possess some etiological relationship. Prominent among

¹ Tractatus de rachitide, etc., Lond., 1650. Ref., Rehn, Gerhardt's Handb. d. Kinderk., III, 1, 43.

² Journ. Amer. Med. Assoc., 1900, March 24.

³ Ergebn. d. inn. Med. u. Kinderh., 1909, IV, 437.

⁴ Jahrb. f. Kinderh., 1884, XXII, 118.

⁵ Scottish Med. and Surg. Journ., 1905, XVI, 231.

⁶ Jahrb. f. Kinderh., 1896, XLII, 273.

⁷ Arch. f. Kinderh., 1888, IX, 293.

these is *diet*. It is certain that the disease is much less frequent and less severe in breast-fed children unless nursing is unduly prolonged, and it has been maintained that deficiency in the percentage of fat is the chief cause in artificially fed children, especially if combined with an insufficient amount of proteid matter and an excess of carbohydrates. There is no positive proof of the actual influence of any one of these dietetic factors. It is clear, at any rate, that infants fed upon condensed milk or other proprietary food are especially prone to develop rachitis, these foods generally possessing just the defects mentioned. Yet a deficiency in any of the food-elements is not the only agent, for the disease may follow over-feeding as well as under-feeding. A very great deficiency in the amount of food given leads to wasting without the development of rachitis; and on the other hand, it is not infrequent that babies become rachitic who have been fed entirely upon breast-milk of the best quality. This is especially true of the Italians and Negroes in the northern cities of the United States. Of the cases reported by Morse¹ 18 per cent. were breast-fed solely. If the food-influences are active agents, it is forced upon us that we do not yet know in just what the errors in diet consist.

Hygienic conditions in general seem to be important factors. Over-crowding among the poor in cities, with the attendant lack of fresh air and sunlight and other disadvantages, certainly predisposes, and the disease is less common in country districts. Yet, on the other hand, rickets is common, although generally less severe, among the well-to-do, where both the hygienic surroundings and the diet have been carefully supervised. I have repeatedly seen it develop in infants under the best conditions in spite of every precautionary measure.

Season is of influence, the disorder being less frequent during the summer and sufferers from it seeming to improve at this time. This common clinical observation has been confirmed by the autopsy-statistics of Schmorl² upon 281 cases of rickets. The microscopic study of the bones showed evidences of a beginning rachitic process in comparatively few cases during the summer; while the proportion of those with evidences of healing was at its maximum at this time. *Geographical distribution and climate* are likewise factors. The disease is especially frequent in northern temperate climates, less common in subtropical countries, and rare in the tropics and in the far north. In England, Austria, Northern Italy, the Netherlands and Germany it is very common, while it is relatively rare in China, Japan, Greenland, Turkey, Denmark, Norway, Greece, Southern Italy and Southern Spain (Palm).³ It is less common at high altitudes than at lower elevations in the same country. The lesser frequency in summer-time or in hot countries probably depends, at least to some extent, upon the greater open-air life. In the tropics, also, breast-feeding is more usual.

Apart from locality, *race* seems to exert a powerful influence under certain conditions. Rickets is particularly prevalent in races from warm regions transported to colder ones. Thus among the Italians and Negroes of the northern cities of the United States nearly every infant exhibits more or less rickets, while in the southern United States it is far less common among the Negroes, and rare in these in the tropics, and it is not frequent in southern Italy. This racial prevalence in northern cities cannot be the result of improper dietetic or hygienic conditions, for these

¹ Bost. Med. and Surg. Journ., 1899, CLX, 163.

² *Loc. cit.*, 439.

³ Practitioner, 1890, XLV, 270.

are in no way inferior to those active among the poor immigrants from other countries. This brings up, naturally, the question of *heredity*. Although formerly denied, certain more recent investigations decidedly support the view that the tendency to become rachitic, once having developed, is readily perpetuated and increased by inheritance, and this seems to be the most satisfactory explanation of the remarkable race-disposition alluded to. Siegert¹ especially has emphasized this fact, and I have myself seen instances strongly corroborative; and Zimmern² and others have reported cases showing remarkable family tendency. There is, however, much against the theory and any decision must be held in abeyance.

Age as a factor is important. Rickets is observed especially in the first 2 years of life. It rarely shows itself clinically before the 3d month and generally not until after the 6th month. Baginsky,³ in 620 cases found symptoms present in 43 from 3 to 6 months of age; 173 from 6 to 12 months; 220 from 12 to 18 months; and 113 from 18 to 24 months. It rarely begins after the 2d year of life. Its height is usually about the end of the 1st or the beginning of the 2d year. Even cases of *fetal rickets* have repeatedly been reported, but doubt attaches to most of these. (See *Fetal Rickets*, p. 598.) So, too, *late rickets* has been described, the symptoms not appearing until later childhood or puberty. The condition is, however, certainly very rare and does not differ essentially from osteomalacia. (See *Late Rickets*, p. 598.)

The condition of the parents, apart from the existence of rickets in them, already referred to, predisposes to the development of the disease only by the general debilitating effect upon the offspring or through the production of poor breast-milk by the mother. *Sex* exerts no influence of moment. *Previous disease of the infant* has a definite influence. Thus, prolonged catarrhal states of the digestive apparatus may be followed by the disease, perhaps through rendering the child unable to profit by food which, in itself, is of good quality; perhaps through favoring the production of some toxic substance in the intestine. Should the digestive disturbances be severe, however, wasting of the child is liable to occur rather than rickets, and, on the whole, digestive disorders are not a very prominent etiological factor. Acute severe disease of any nature appears sometimes to be the starting point for rickets, or to make the symptoms worse if already present.

Pathogenesis.—The relationship of the various etiological factors to the pathological process resulting is not yet understood, and various theories have been advanced. One is that the process depends upon an insufficient amount of mineral matter in the food. For this there appears no good evidence, since the artificial food given to infants is in no respect less well provided with mineral matter than is human milk. The experimental feeding of young animals with food deficient in lime has produced a condition resembling osteoporosis, but not the true lesions of rachitis (Schabad).⁴ Another theory advanced is that the lime-salts in the food may be sufficiently abundant, but that there is imperfect absorption of these from the intestinal tract. This has been proven by Rüdell⁵ to be incorrect. The view has likewise been expressed that the disease is

¹ Jahrb. für Kinderheilk., 1904, LVIII, 237.

² Nouvelle iconog. de la Saltpetrière, 1901, XIV, 299.

³ Prakt. Beiträge z. Kinderheilk., 1882. Ref. Vierordt, Nothnagel's Spec. Path. u. Therap.; Rachitis, VII, 2, 1.

⁴ Arch. f. Kinderh., 1909, LII, 47.

⁵ Arch. f. exp. Path. u. Pharm., 1894, XXXIII, 91.

produced by acid in the blood which removes the lime-salts from the system. All theories involving an increased excretion of lime-salts or a failure in the supply of these to the bones as the cause of rickets are negatived by the fact demonstrated by Stöltzner¹ and by Brubacher² that the other tissues of the body contain a normal amount of this substance. It should be stated, however, that Aschenheim and Kaumheimer³ do not agree with this conclusion. That the calcium and phosphorous balance is disturbed in rickets has been shown by Schabad⁴ and others, the excretion of both phosphorus and calcium being increased in the acute stage and decreased during convalescence. It seems certain that the bones are unable to retain a normal amount of calcium.

That improper diet is instrumental in producing the disease in young, wild animals in confinement seems indicated by various reported experiments. The results, however, are inconclusive, since animals under these conditions appear predisposed to rickets even when fed in a proper manner. The exact influence of diet is still unsolved. The theory of the infectious nature of the disease has been advocated by a number of observers, but has not obtained general recognition. Yet the great prevalence of rickets in certain localities, and the comparative absence of it from others where dietetic and hygienic conditions do not appear to be any more favorable, renders it possible that this may be the solution.

Rickets would appear, according to some views, to be essentially an inflammatory process in the bones, although how this is brought about is unknown, and even the existence of inflammation at all is denied by other writers, who regard the disease rather as an interference with development, the result of the action of some irritant. Whether this substance is a toxic material from the intestinal canal, an abnormal internal secretion, or some other as yet unsuspected agent, is not known. The influence of various of the internal secretions upon the production has been maintained, but has not as yet been proven.

We can only conclude, that in some way in rickets there occurs a failure of lime-salts to be deposited through some inability of the bones to absorb it, a cessation in the transformation of osteoid tissue into true bone; but of the manner in which this is brought about we are entirely in ignorance. The disease is a nutritional one, in which diverse debilitating factors appear able to disturb in some way the normal nutritional balance of the infant, and imperfect metabolic processes result.

Pathological Anatomy. Lesions of the Bones.—The principal anatomical lesions are in the osseous system. Macroscopically the bones are softer and more flexible than normal, the degree to which they may be bent being remarkable in severe cases. All the bones of the body are affected but the changes are especially marked in the long bones. There is enlargement at all the epiphyseal junctions, but most decided at the wrists and ankles and at the costochondral articulations (Figs. 195, 196 and 197), the thickening in the latter position being always much more evident on the pleural surface. Moderate thickening of the shaft of the long bones also takes place and all the distinctions of outline marked by angles, ridges and the like, are less marked than in normal bones. There is also thickening of the flat bones in certain regions, with the production, especially upon the skull, of large bosses; while in other portions of the

¹ *Jahrb. f. Kinderh.*, 1899, I, 268.

² *Zeitschr. f. Biol.*, 1890, XXVII, 517.

³ *Monatsschr. f. Kinderh., Orig.*, 1911, X, 435.

⁴ *Arch. f. Kinderh.*, 1910, LIII, 380; LIV, 83.

cranium spots of very thin, parchment-like bone may be found (Cranio-tabes). (See Vol. II, p. 428.) These alterations produce deformities of many sorts, and fractures are common (Fig. 198). The degree of deformity varies greatly, depending upon the severity of the case. Arrested growth in the length of the bones may occur as a result of the changes in the epiphyses.



FIG. 195.—RADIOGRAPH OF THE WRIST IN RICKETS.

Shows the enlargement at the wrist-joint. From a colored child, aged 2 years and 9 months, in the Children's Hospital of Philadelphia.

Longitudinal section shows the junction of the shaft and the epiphyses much wider, thicker and softer than normal (Fig. 199) with a broadening of the bluish, cartilaginous layer. The centres of ossification are hyperemic, spongy and enlarged. It is to this and to the general widening of the cartilaginous layer, that the enlargement of the epiphysis is due. The medulla of the shaft is redder and more jelly-like than normal and the medullary cavity broadened and its bony divisions rarefied. The periosteum of both the flat and long bones is hyperemic, and beneath it are

several layers of friable, spongy, vascular tissue, with large intertrabecular spaces.

Microscopically there are very characteristic changes seen. The proliferating layer of the epiphyseal cartilage, situated next to the hyaline



FIG. 196.—ANATOMICAL SPECIMEN OF RACHITIC CHEST.

Shows the rachitic rosary produced by enlargement at the costo-chondral articulations.

cartilage, is hyperemic and the cartilage cells more numerous than normal and irregularly arranged. The columnar zone next to it is still more affected in the same way, being abnormally wide and exhibiting an unusual length of columns of cells and increase of vascularity. This



FIG. 197.—RACHITIC CHEST VIEWED FROM THE INSIDE.

Same case as in Fig. 196.

is not due to an abnormal production of cartilage but to a failure of it to be transformed into bone in the normal manner (Schmorl).¹ The layers of cartilage are transversed irregularly by canals containing much-dilated blood-vessels and soft, vascular, imperfectly formed "osteoid" tissue, deficient in lime-salts. The zones of calcification and, last, of ossification,

¹ *Loc. cit.*, 424.

are broader than in healthy bone and have lost their normal, sharply-defined outlines, exhibiting instead calcified areas irregularly mingled with others still cartilaginous, and with scattered, unusually large medullary spaces containing osteoid tissue. This disappearance of the sharply-defined junctions of the zones, as seen in the normal epiphysis, with the irregular inroads of one into the other, is one of the most striking characteristics of rickets. The spongy portion of the shaft exhibits increased vascularity, with erosion of the trabeculae, resulting in large, medullary spaces.



FIG. 198.—SKELETON OF A SEVERE CASE OF RICKETS.

From a negro child of 6 years, in the Pennsylvania Hospital of Philadelphia at the time of his death. (*Donhauser, Bull. of Ayer Clinical Laboratory, 1907, No. 4, 13.*)

Beneath the periosteum of both long and flat bones an analogous process is seen, with cell-proliferation, vascularity, and imperfect calcification, resulting in thickening and in the production of osteoid tissue.

Chemically the rachitic osseous tissue is very decidedly altered in the amount of lime and of phosphorus contained, these being sometimes diminished to 50 per cent. or even 25 per cent. of the normal. The percentage of organic matter and of water is increased. These characteristics account for the unusual degree of softness and flexibility of the bones, which suffer in consequence from green-stick fractures, with the production of a large amount of callus.

During recovery from the disease the bony changes disappear through resorption and condensation of the imperfect bone taking place, and lime-salts are again deposited. The development of very hard, compact, dense, bony tissue (eburnation) results.

In brief, the osseous changes in rickets consist in unusual hyperemia; excessive proliferation of cartilage; irregularity in the transformation of cartilage into bone, with disturbance of the normal division into zones; deficient deposit of lime-salts, with consequent over-development of osteoid tissue which fails to change into true bone; thickening of the periosteal layer through undue cellular production, and the formation of masses of osteoid tissue here; absorption of trabeculæ in the spongy bone, with consequent increase of the medullary spaces; and hyperemia and widening of the central marrow-cavity.

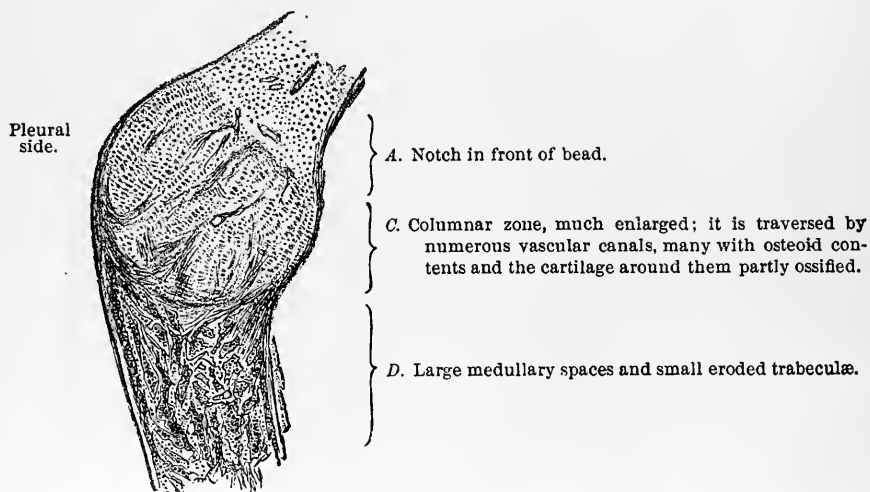


FIG. 199.—LONGITUDINAL SECTION OF RIB OF RACHITIC CHILD AGED 15 MONTHS.

About 3 times natural size. (*Barlow and Bury, Keating's Cyclopædia of the Diseases of Children*, 1889, II, 241.)

Lesions of the Soft Parts and Organs.—The spleen is often enlarged, yet not necessarily so. Catarrhal conditions of the lungs are frequent, with areas of atelectasis and others showing emphysema. Moderate enlargement and fatty infiltration of the liver may occur. The gastro-enteric tract often exhibits catarrhal inflammation with unusual distention of the stomach and intestines. The lymphatic glands are frequently enlarged. Thinness and weakness of the muscles are evident; well seen in the abdominal walls. The ventricles of the brain are often moderately dilated.

Symptoms.—Although the skeletal changes are the most characteristic, the constitutional symptoms are the first to become manifest. These seldom are noticeable before the age of 3 months, although the microscopical changes in the bones appear even in the 2d month. Among the earliest manifestations are head-sweating, restlessness, and rocking of the head when asleep. These early symptoms develop gradually, and usually attract little attention. In a short time beading of the ribs is noticed in nearly all cases. Other bony enlargements now develop, especially of the skull, ribs, wrists, and ankles, while character-

istic alterations of the shape of the chest appear and the abdomen becomes pot-bellied. The general health may remain unaffected at first, but in well-marked cases the child finally grows anemic and suffers readily from respiratory and gastrointestinal catarrhal processes and sometimes convulsions. The teeth are late in appearing and decay early; the fontanelle fails to close at the proper time; growth of the body is interfered with; and when sitting and walking begin, at a period much later than normal, curvature of the long bones and of the spine is produced.



FIG. 200.

FIG. 200.—RACHITIS, SHOWING ESPECIALLY THE LATERAL DEPRESSION OF THE THORAX AND CURVATURE OF THE TIBIÆ.



FIG. 201.

FIG. 201.—RACHITIS, SHOWING THE POT-BELLY AND THE DISTORTION OF THE EXTREMITIES
From a child of 4 years, in the Children's Medical Ward of the Hospital of the University of Pennsylvania.

The complex of symptoms and the general appearance of the patient are very characteristic (Figs. 200 and 201) and the diagnosis easy. A more detailed description is required:

Head.—The head appears, and often is, larger than normal in its horizontal circumference and is usually brachycephalic. The forehead is prominent, and the occiput and vault flattened. This produces a peculiarly box-like form (Fig. 202). The shape, which develops oftenest about the end of the 1st year, depends largely upon the deposit of rachitic bone which takes place especially upon the frontal and parietal eminences, along the sutures, and about the fontanelles. The flattening of the occipital region is due to pressure upon the pillow; and should the child while in bed lie very constantly on one parietooccipital region, great asymmetry of the head may result, one oblique diameter being much shorter than the other, one ear nearer the front, and even one cheek more prominent. The anterior fontanelle in rickets is larger than normal, and its time of closing

is delayed, occurring sometimes not until after the 2d year. In severe cases the posterior fontanelle and the sutures remain open until the end of the 1st year. In the posterior portion of the skull thin, soft membranous spots are often found in the 1st year (craniotabes, Vol. II, p. 428). The veins of the scalp are distended, especially over the temporal regions. The hair is quite commonly worn away from the back of the head by the frequent rocking movements. The face seems small, the upper jaw being narrower than normal and the lower somewhat square. These various deformities usually disappear as recovery takes place, but the relative increase in size may persist in severe cases to some extent throughout life. The teeth usually appear late and out of the normal order, not infrequently none having erupted during the 1st year. They often decay early, and have a tendency to exhibit furrows.



FIG. 202.—MODERATE RACHITIC ALTERATION OF THE HEAD.

Shows the prominent frontal bosses, in a patient, aged 9 months, in the Children's Hospital of Philadelphia.

Thorax.—Beading of the ribs at the costochondral articulations is the most frequent and the earliest osseous symptom. It is quite visible to the eye unless the infant is well covered with fat. Morse¹ found it in all of 318 cases of rickets, and the only symptom in 41 per cent. of these. In all well-marked cases there is in addition a depression of the cartilages anterior to the epiphyseal enlargements. A flattening or even a concavity of the sides of the thorax in a vertical direction develops, extending backward to the posterior axillary line and downward about as far as the 7th rib. As a result of this alteration the sternum is unusually prominent, producing a condition suggesting "pigeon breast," but usually differing somewhat from the typical instances of this seen in certain other conditions, in that the whole sternum is pushed forward in a straight line, without any anterior curvature of it. The rachitic thorax also exhibits bulging at the angle of the ribs posteriorly, accompanied by a flattening of the back. These changes produce in well-marked cases a deformity known as the "violin-shaped" chest.

¹ Journ. Amer. Med. Assoc., 1900, March 24.

In addition to the vertical lateral depression there is a horizontal one (Harrison's groove) caused by the decided flare of the costal border. This is at about the level of the xiphoid cartilage and corresponds to the insertion of the diaphragm. Occasionally a funnel-shaped depression of the lower part of the sternum occurs. In some cases the clavicles are thickened at the extremities, shorter, and more curved than normal, and may show green-stick fractures.

The thorax as a whole is lengthened in an anteroposterior direction, and is small and narrow, except for the sudden widening of the lower



FIG. 203.—MULTIPLE DEFORMITIES IN A SEVERE CASE OF RICKETS.

From a child in the Children's Ward in the University Hospital, Philadelphia. Shows especially the great contraction of the thorax with the costal flare.

portion (Fig. 203). The deformities are produced by the action of the muscles of respiration, including the diaphragm, upon the very soft bones; and by the pressure of the back against the bed, and of the distended stomach and intestines and the enlarged liver against the costal border.

Spinal Column.—This may be normal, but a certain degree of lateral curvature is common in infants, and may be very persistent. Still more frequent, and always seen in well marked cases, is a long posterior curvature dependent upon the weakness of the ligaments and of the

muscles of the trunk. It usually disappears entirely or to a large extent when the child is suspended from the arm-pits (Figs. 204 and 205).

Pelvis.—In severe cases the pelvis may become permanently deformed, the anteroposterior diameter being shorter and the outlet narrower, with thickening of the crests of the ilia.

Extremities.—Epiphyseal enlargement at the wrist (Fig. 201) is an early symptom nearly always present, and that of the ankle is also very frequent. Similar enlargement is often discoverable at the lower end of the humerus and femur, less frequently at the upper end of these bones and of the tibia and fibula. Bending of the shafts of the long bones occurs in the lower extremities, the commonest deformities being knock-knees and bow-legs (Fig. 206). (See Vol. II, pp. 424, 425.) The tibia may be bent outward or forward (Fig. 207). The femur is curved



FIG. 204.—RACHITIC SPINAL CURVATURE.

Very well marked when child is sitting. *Courtesy of Dr. H. R. Wharton.*

only in the severe cases, and generally in a forward and outward direction. Coxa vara is another rachitic deformity. Sometimes a striking deformity results from the child sitting cross-legged, the femora being partly rotated outward and the tibiae and fibulae fitting into each other where pressure one upon the other has been constantly exercised. Bending of the humerus is not common but the bones of the forearm often curve outward. Greenstick fractures on the concave side of the bone are not infrequent, especially in the forearm and the tibia, and add to the deformity. Bending is produced largely by the action of position and of muscular pull upon the soft bones, although partly by asymmetrical growth of the epiphysis. It occurs in the legs even before walking is commenced, but is much increased by it. The deformity of the extremities becomes a marked symptom toward the end of the 1st year.



FIG. 205.



FIG. 206.

FIG. 205.—RACHITIC SPINAL CURVATURE.

Same case as in Fig. 204. Very decided diminution in the degree of curvature when the child is suspended. *Courtesy of Dr. H. R. Wharton.*

FIG. 206.—SEVERE CASE OF RICKETS.

Illustrates the enlargement of the wrists and the curvature of the legs. *Courtesy of Dr. H. R. Wharton.*



FIG. 207.—RACHITIC DEFORMITY, SHOWING THE CURVATURE OF THE LIMBS AND THE WELL-MARKED POT-BELLY.

Patient in the Children's Ward of the University Hospital, Philadelphia.

Growth of the long bones in length is very commonly interfered with, especially in the lower extremities, and in the worst cases permanent dwarfism may be a final result. Occasionally thickening of the fingers occurs. Flat-foot is a common rachitic deformity.

Ligaments.—Relaxation of these aids in producing deformity. It is seen especially in the spine and in the larger joints, aiding in the production of knock-knee, over-extension of the knee-joints, weak ankles, and scoliosis.

Muscles.—Lack of tone of the muscles of the body is a symptom commonly present. They are poorly developed, pale, and small. As a result, cases of well-marked rickets are very late in standing and walking, this being occasionally deferred even until early childhood is well under way. In one instance a child of 4 years was unable to walk, although without any serious deformity. The lack of muscular power is often



FIG. 208.—RACHITIS, WITH DIASTASIS OF THE RECTI ABDOMINIS.

Shows displacement of the intestines through the split in the muscles. Colored child of 15 months. (*Francine, Arch. of Pediat.*, 1904, *Feb.*, 116.)

indeed so great that the diagnosis of paralysis may be made. I have seen the mistake occur repeatedly. The condition of "pot-belly," a nearly constant symptom of rickets, depends to a large extent upon weakness of the abdominal muscles, although the weakness of the gastric and intestinal walls, combined with the tendency to flatulent distention, aids in its production. The abdomen is uniformly distended, tympanitic, and not tender on pressure. Very commonly in marked cases there results a diastasis of the external recti of the abdomen, well shown when the child attempts to raise itself from a recumbent position (Fig. 208). Constipation depends upon the muscular weakness of the intestinal and abdominal walls.

General Condition.—The skin is usually pale and transparent, with a tendency to enlargement of the veins in many localities, especially over the scalp and at the root of the nose. Although often apparently well nourished, so far, at least, as the presence of adipose is concerned, the tissues of children with rickets are nearly always flabby. Severe cases

eventually lose considerably in weight and in general health and become anemic. The general resisting power is slight.

Digestive System.—Adenoid growths and tonsillar hypertrophy are common. The sensitiveness of the mucous membrane is shown by the readiness with which rachitic subjects develop obstinate catarrhal disturbances of the stomach and intestines from slight causes. This aids in producing the tympanitic distention of the stomach and intestines and the characteristic pot-belly referred to. Constipation is common, often alternating with diarrhea. The liver is not infrequently enlarged, but this may be only apparent, due to the displacement downward, the result of the distortion of the chest.

Respiratory System.—The mucous membranes in rickets are particularly prone to the development of diseased conditions, among them bronchitis and bronchopneumonia. Acceleration of respiration, without fever or other discoverable cause, and often accompanied by moving of the alæ nasi, is very characteristic. The breathing in well-marked cases is largely diaphragmatic, due to the softening and yielding of the framework and the weakness of the muscles of the thorax. The deformities of the chest produce alterations in the physical signs of auscultation and percussion, which may be very misleading in the diagnosis of possible intra-thoracic diseases.

Circulatory System.—In mild cases the blood is unaffected, but in the severer ones more or less anemia usually develops. Morse's¹ studies of his own cases and of the writings of other investigators led to the conclusion that the blood in rickets usually shows the red cells normal or reduced in number, and the hemoglobin always reduced to a greater degree. Leucocytosis is present in about half the cases. Dilatation of the superficial veins is decided.

Nervous System.—Nervous symptoms are very common, prominent among them being restlessness at night, with rocking of the head upon the pillow, and constant tossing off of the bed-clothes. The unusual instability of the nervous system characteristic of rickets would appear to predispose to convulsive conditions of various sorts, as tetany, laryngospasm, and particularly eclampsia. These symptoms are those, however, of spasmophilia, which seems in many instances to be associated in some way with rickets. See Spasmophilia, Vol. II, p. 249.) In a study of 1766 rachitic children, Oliari² found evidences of spasmophilia in 542. Pain in the bones, especially evident when the infant is lifted by grasping the chest, is sometimes seen. Although perhaps due in some cases to the rachitic lesions, it is probably oftener dependent upon a complicating scorbutus. In the category of nervous affections may be placed the profuse sweating of the head, which occurs during sleep irrespective of the weather, and which is sometimes sufficient to moisten the pillow. Fever may be present, but does not appear to be a symptom of the disease. In severe cases there may be decided mental backwardness associated with the general weakness.

Urine.—No special alterations are discoverable.

Glandular System.—The external lymphatic glands are frequently found enlarged. Moderate enlargement of the spleen is common and great increase in size occasional, but it is still undetermined whether this hypertrophy is a symptom of rickets or only a complication. It must be remembered, too, that the wide costal flare so often present, and the

¹ Journ. Amer. Med. Assoc. 1900, March 24.

² La Pediatria, 1910, XVIII, 581.

diminished capacity of the thorax, renders the spleen much more readily felt.

Fetal Rickets.—Whether or not such a condition as fetal or congenital rickets exists has been much disputed. It is claimed as very common by some, but denied absolutely by other investigators. Certainly it is rare in the United States; and it is, moreover, probable that the majority if not all of the reported cases are instances of osteogenesis imperfecta or of chondrodystrophy fetalis (see Vol. II, pp. 433 and 428), which differ pathologically from the rachitic process. An elaborate review of the subject by Wieland¹ leads him to the conclusion that not a single case beyond criticism has been reported.



FIG. 209.—LATE RICKETS.

Patient 28 years of age, in whom the rachitic changes began at the age of 8 years. Photograph taken at the age of 21 years. (von Bökay, *Arch. de méd. des enfants*, 1910, XIII, 444.)

Acute Rickets.—Although cases of this condition have been described in which the symptoms develop with great rapidity, it is doubtful whether this condition is not something entirely different from rickets—in many cases certainly infantile scurvy. Other cases are, perhaps, only instances of rapid development of rickets in a severe form, but this cannot properly be called acute.

Late Rickets.—The pathological processes of osteomalacia are so similar in some respects to those of rickets, although the causes are different, that it is probable that the instances of so-called late rickets, developing in later childhood or at puberty, are for the most part properly to be classified as early osteomalacia. Schmorl² and others, however, from a careful study of the subject maintain that genuine rickets may develop in later childhood. The condition is certainly rare. Its devel-

¹ *Jahrb. f. Kinderheilk.*, 1908, LXVII, 675; *Ergebn. der inn. Med. u. Kinderh.*, 1910, VI, 64.

² *Arch. f. klin. Med.*, 1905, LXXV, 170.

opment is usually slow, the course prolonged, and the final deformities very decided (Fig. 209).

Complications and Sequels.—The great tendency to the development of respiratory affections, especially bronchitis and bronchopneumonia, has already been alluded to. Atelectasis not infrequently occurs in infants with severe rachitic deformities of the chest. The gravity of all respiratory diseases in rachitic subjects is greatly increased by the yielding character of the thoracic walls and the insufficient expansion of the lungs. The disposition to diarrhea or constipation has also been mentioned. General convulsions are common, as are laryngospasm and tetany. This tendency to the development of spasmophilic symptoms, particularly convulsions, is especially marked in rickets. Umbilical hernia is of frequent occurrence, due to the distention and thinning of the abdominal walls. Infantile scurvy usually manifests itself in subjects already rachitic, although to this there are frequent exceptions. Bony deformities of various sorts remain as sequels, sometimes permanent.

Course and Prognosis.—Rickets is a chronic but self-limited disease. The active symptoms usually continue not longer than the beginning of the 1st and seldom more than the end of the 2d year, their retrogression being evidenced by the gradual spontaneous cessation of head-sweating; the improvement of the general restlessness, irritability, and other nervous symptoms; disappearance of anemia; closure of the fontanelles; and cessation of the disordered intestinal and respiratory states and of the softness of the bones. Recovery from the bony deformities is very slow. In most cases the enlargement of the epiphyses will finally almost entirely disappear, as will the deformity of the skull. Sometimes, however, the head remains always larger than normal. Even moderate bowing of the legs and other deformities disappear spontaneously in a remarkable manner. Recovery is usually complete by the end of the 3d or 4th year. In well-marked cases of the disease, however, permanent deformities are likely to be present throughout life, in the form of bow-legs, knock-knees, curvature of the bones of the thigh, leg, or forearm, deformities of the chest, scoliosis, flat-foot, rachitic pelvis, rachitic coxa vara, and dwarfing from arrested growth of the bones in length.

The disease in itself is not dangerous to life, but through its numerous complications is the cause of many deaths. The contraction of the chest and the lack of resiliency in its walls are very serious factors should pneumonia or pertussis be contracted, and severe cases of rickets may die from ordinary bronchitis through the development of atelectasis. The debility and general loss of resisting power predispose to a fatal ending in cases of intestinal disease. Convulsions attending rickets are the cause of death in numbers of instances.

Diagnosis.—Well-developed rickets is not readily confounded with any other disease. Early in its course it is not so easily recognized. The principal early diagnostic symptoms are the head-sweating, and the restlessness at night. Later are evident the enlarged fontanelles, characteristic shape of the head, distended abdomen, alteration in the shape of the chest, rachitic rosary, and the various other deformities described.

Certain other conditions occasion difficulty in diagnosis. *Hydrocephalus* has a superficial resemblance to the rachitic head in some instances. In both the fontanelles are large and the sutures open, but in *hydrocephalus* the head is of a more globular shape and the sides protrude somewhat beyond the ears, while in rickets the shape is more rectangular.

with areas of decided thickening of the bones, especially over the parietal and frontal eminences and about the fontanelles. *Craniotabes* is not positive diagnostic evidence of rickets, as it may occur in syphilis also.

Delayed dentition, although a diagnostic symptom of rickets, is not necessarily so. If rickets is somewhat late in developing, the first teeth may erupt promptly; and, on the other hand, it is not infrequent for entirely healthy infants to have no teeth until the age of 10 or 11 months, or even a year. *Infantile scurvy* has often been confounded with rickets because the two are so frequently combined; and the names "scurvy-rickets" and "hemorrhagic rickets" have been applied to infantile scurvy. Either disease can occur without the other, and the two have, in reality, no symptoms in common. The tenderness of the bones which sometimes seems to be a symptom of rickets is, in most cases, as already pointed out, probably due to a certain scorbutic element. *Infantile osteomalacia* has been described, but is to be considered identical with rickets in most instances, although probably some of the cases reported belong to the category of *osteogenesis imperfecta*.

Osteogenesis imperfecta has often been called "fetal rickets." In it there is usually associated a remarkable thinness of the flat bones, especially of the skull, with more or less deformity of the long bones. It has little in common with rickets. *Osteopsathyrosis*, or *fragilitas ossium*, has likewise often been confounded with rickets. It is true that in severe rickets there is a decided tendency to the occurrence of fractures, yet these are usually of the green-stick variety, due to softness of the bone. In true *fragilitas ossium* there is nothing of the soft character present, and neither chemically nor microscopically is there anything found resembling rickets. The two conditions are absolutely distinct, as I have pointed out elsewhere,¹ and *osteopsathyrosis* is more closely associated with some forms of *osteogenesis imperfecta*.

Infantile myxedema in its early stages bears certain resemblances to rickets. In both there is delay in dentition and in the closing of the fontanelles, and slowness in learning to walk. In cretinism, however, there is an unusual slowness of growth in length, and the peculiar physiognomy of the disease, with mental impairment and the general physical characteristics, soon make the diagnosis easy. *Paralytic conditions* of various sorts, with wasting of the muscles, sometimes lead to the suspicion of rickets. In all the learning to walk may be long delayed, but in none are the other symptoms of rickets present; while careful study may show the characteristic electrical reactions and other evidences of poliomyelitis, or the spastic condition of a cerebral paralysis.

Syphilis has little in common with rickets. The syphilitic pseudo-paralysis occurs usually in the early months of life, antedating the weakness of rickets, and the sabre-tibia of later syphilitic manifestations is dependent solely upon a thickening of the anterior border of the bone and is unaccompanied by other conditions suggesting rachitic deformities. The spinal curvature of *Pott's Disease* is short and angular in shape, in contra-distinction to the long anteroposterior curve of rickets, and when well-marked does not disappear when the child is lifted by the arms.

Treatment. Prophylaxis.—In the absence of exact knowledge regarding the cause, prevention is difficult. Certainly the most favorable hygienic conditions possible should be obtained, including abundance of fresh air and sunlight. The use of proprietary amylaceous foods and

¹ Amer. Journ. Med. Sci., 1897, April.

of condensed milks should be avoided. Breast-feeding is to be employed whenever possible. That cod liver-oil is of benefit after the disease has developed indicates that it might be of service as a prophylactic measure; and Hess and Unger¹ tried it with satisfactory results in a Negro community, finding that the disease failed to develop in 80 per cent. of the infants who received the treatment.

Treatment of the Attack.—This should be commenced as early as possible. Defects in *diet* should be sought for and removed, as much care being taken to avoid overfeeding as underfeeding. Dietetic changes are necessarily, to an extent, experimental in this disease; but some alteration should be tried, even although the infant seems to be thriving in other respects. If the feeding is manifestly faulty, it should, of course, be corrected in a way to accord with the needs of the patient. (See Feeding, p. 108.) The use of proprietary infants' foods should be stopped. If amylaceous foods have constituted a large proportion of the dietary, these should be abandoned largely for a time. Possibly an increase of the fat in the food may be of service, if well tolerated by the digestion. Beef-juice, orange-juice, cooked fruit, and broths may be added to the diet. Sometimes scraped or minced underdone meat is of advantage. Fresh vegetables must be tried as soon as the age permits. If no cereal food has yet been given, its administration may be begun. In fact, a varied diet is an excellent means for the cure of rickets; and this should be commenced earlier than is commonly done with normal children; continuing milk at least in large enough amount to supply sufficient calcium to the organism. Caution must be used in making any change, not allowing it to be too sudden or too radical, lest other evils than rickets follow.

All other diseased conditions, especially digestive disturbances, must be remedied as far as possible. *Hygienic treatment* is important, particularly life in the open air, massage, cool baths when well borne, and change of residence to the seashore or country. *Medicinal treatment* of various sorts has been employed. General experience has shown cod-liver oil to be of unquestionable benefit. It is usually well borne except in the hottest weather, and even then may be given tentatively. Phosphorus in doses of $\frac{1}{300}$ or $\frac{1}{200}$ grain (0.0002 or 0.0003) 3 times a day, has been especially recommended by Kassowitz,² and used very extensively by others, but its value is very doubtful in the opinion of many. The experience of Schabad³ indicates that although phosphorus exerts no influence upon the calcium-metabolism in normal infants, yet when combined with cod-liver oil it favors the retention of calcium in cases of rickets. This supports the clinical experience of the value of the combination of phosphorus with cod-liver oil as reported in many quarters. Lime in various forms was formerly much given, on the ground that a deficiency of lime in the food produced the disease. It is now well recognized by most authorities that this theory is incorrect, and that the administration of lime can have no direct influence upon the rachitic process. There is sufficient lime in the usual food of infancy. It is only during convalescence, when special demands may exist for an unusually large amount to replace the deficiency which has developed, that the administration of lime may be of possible value. It may be given in the form of the citrate or lactate. Iron, and sometimes

¹ Journ. Amer. Med. Assoc., 1917, LXIX, 1583.

² Wien. med. Blätter, 1883, VI, 1492.

³ Zeit. f. klin. Med., 1909, LXXVIII, 94; 1910, LXIX, 435.

arsenic, are of advantage when anemia is marked, and general tonic treatment should be given when indicated. Various organs and tissues have been used therapeutically for rickets, among them thymus gland (Mettenheimer),¹ thyroid gland (Heubner,² and others), suprarenal body (Stöltzner)³ and bone-marrow (Amistani).⁴ The value of none of these has been established.

The development of *deformities* must be carefully guarded against. Rachitic children should be discouraged from walking until the bones have become firm. The wearing of too thick a diaper predisposes to the development of bow-legs. Carrying the infant always on the one arm is very likely to produce scoliosis. Sitting cross-legged may occasion rotation of the femora, indentation of the tibiæ, and curvature of the forearms from pressure of the hands against the bed or floor. Irregular distortion of the head is to be avoided by altering the position in which the child lies. The treatment of deformities already acquired is considered fully in textbooks upon orthopedic surgery. Here only may be mentioned the very great value of massage in strengthening the feeble muscles; the importance of favoring free movement of the limbs by the patient by creeping and by any other form of exercise, properly regulated, which does not directly increase deformity; and the correction of deformities by postural treatment and the like, such as lying upon one side with a pillow under the kyphotic spine; upon the abdomen; the gentle bending of curved extremities by the nurse; etc., etc., according to the nature of the deformity present. Such measures, combined with general treatment, are often completely efficacious without operative or special orthopedic procedures. By the age of 3 years, however, and often decidedly before this, the ossification of the bones has become too complete to permit of benefit being obtained in this way.

CHAPTER II

SCORBUTUS

(Infantile Scurvy)

This disease as seen in early life does not differ materially from that in adults, long known as occurring among sailors and others deprived of suitable food. The adjective "Infantile" is not strictly correct, since the disorder is occasionally observed in children after this period. In infancy it was first described by Möller,⁵ but considered by him to be "acute rickets." Barlow,⁶ in 1883, made a careful study of it and recognized its true nature, and the disease has often been called after his name. Numerous contributions to its literature have since been made, one of the most extensive being the Collective Investigation of the American Pediatric Society in 1898,⁷ in which I was actively interested.⁸ An able review of the subject has been published by Concetti,⁹ based

¹ Jahrb. f. Kinderh., 1898, XLVI, 55.

² Berl. klin. Woch., 1896, XXXIII, 700.

³ Path. u. Therap. d. Rachitis, 1904.

⁴ La Pediatría, 1903, XI, 560.

⁵ Königsberger med. Jahrb., 1856-7, I, 377; 1862, III, 135.

⁶ Med.-Chir. Transac., LXVI, 1883, 159.

⁷ Transac. Amer. Ped. Soc., 1898, X, 5.

⁸ Chairman of the Committee.

⁹ Archiv. f. Kinderheilk., 1909, I, 174.

upon these cases with others collected from medical literature, equalling 682 in all. A study of 93 personal cases is given by Morse,¹ and series have been published by others. My own experience is based upon somewhere in the neighborhood of 100 cases. The statistics which follow are taken largely from the report of the American Pediatric Society, in which 379 cases were analyzed.

Etiology.—*Age* is a predisposing cause of importance, the majority of cases occurring in the latter half of the 1st year, and nearly all the remainder before the end of the 2d year. In Concetti's series 359 were between the ages of 6 and 12 months. The youngest case in the Society's report was 3 weeks old. The disease was observed by Owen² in a boy of 12 years. *Geographical distribution* seems to have some influence. Infantile scurvy, although nowhere frequent as compared with other nutritional disorders, appears much more common in some countries, such as England, Germany and the United States, than in others, as France, Italy and Switzerland. This may be either because feeding is more faulty in the regions first mentioned, or very probably to a large degree because the attention of physicians has not been directed to it so closely in the others, and the disease has often escaped recognition. The previous condition of health seems to have no definite influence. About $\frac{1}{4}$ of the cases of the American Pediatric Society's series had previously suffered from some digestive disturbance, while $\frac{3}{4}$ had been in entirely good health. The majority of cases in my own experience, as well as that of others, have occurred in private practice, indicating that matters connected with bad hygiene and social conditions have no predisposing influence, but rather the reverse. The active factor is the employment of an *unsuitable diet* through a period of weeks or months. Just what defect of diet, however, is the agent is uncertain. In 356 cases in the series of the American Pediatric Society, the diet was as follows:

TABLE 78.—INFLUENCE OF FOOD ON THE INCIDENCE OF SCURVY

Breast-milk.....	alone 10 cases, in combination	2—total 12
Raw cows' milk.....	alone 4 cases, in combination	1—total 5
Sterilized milk.....	alone 68 cases, in combination	39—total 107
Pasteurized milk.....	alone 16 cases, in combination	4—total 20
Condensed milk.....	alone 32 cases, in combination	6—total 38
Other proprietary foods.....		total 214

When mixtures of cows' milk were used nothing definite could be learned in the majority of cases regarding the composition of these, which, doubtless, was frequently faulty. Some of the proprietary foods were given with milk, some without. The remarkable influence of change of diet was shown in many of the cases collected by the Committee, and the report justifies certain conclusions, viz.:—That the exciting cause of infantile scurvy is the long-continued employment of an unsuitable diet, but that the fault differs with the individual case, no one diet alone being responsible. Either the needs of the infant appear to demand something wanting in the food, or some harmful element is present. Looking at the cases as a whole it seems beyond question that proprietary infant foods, including condensed milk, are especially liable to produce the disease, and that this is also true of the prolonged heating of milk in a considerable number of instances. It seems entirely unlikely that the cause is a positive one. It is much more probable that the *absence* of some essential

¹ Bost. Med. and Surg. Journ., 1911, CLXX, 504.

² British Med. Journ., 1899, II, 1719.

substance of the nature of a vitamine, as described by Funk,¹ is the etiological factor. Possibly in some instances this is destroyed by the action of heat in the home-preparation of milk, or as employed in the manufacture of the proprietary foods. In those instances of scurvy in which no heating has been employed, there is clearly the absence from the beginning of the necessary element.

Yet in addition, there must exist an *individual predisposing* cause, the nature of which is unknown; otherwise scurvy would be far more common than it is, in view of the vast number of errors of diet existing, and the frequency of sterilization of milk-mixtures. I have seen scurvy occur in several members of the same family during their infancy, and have even observed it in twins, as though there might be a family tendency sometimes existent.

In this connection the production of scorbutus experimentally becomes of interest. This has been successfully accomplished by a number of investigators, among them Holst and Fröhlich² (guinea-pigs; dogs; pigs); Hart³ (monkeys); Ingier⁴ (pigs and guinea-pigs); Fürst⁵ (guinea-pigs); Talbot, Dodd and Peterson⁶ (guinea-pigs and monkeys), all showing that the disease could be produced by the absence from the food of some ingredient, perhaps a vitamine, which may be different in different cases. A one-sided diet, especially if consisting solely of cereals, and in some instances condensed milk (Dodd for monkeys), was employed. The disease was cured by a change in the diet to one of a more mixed character.

Pathological Anatomy.—The principal lesions are those of the bones, combined with a tendency to hemorrhage in various other regions. There occurs a replacement of the bone-marrow by an embryonic connective tissue with few vessels and cellular elements. There is an arrest also in the formation of bone from the osteoblasts, which are few in number, but no production of osteoid tissue devoid of lime-salts such as occurs in rickets (Schmorl).⁷ The bone which is formed is of normal character. The bone-marrow is altered in nature. It loses its lymphoid elements, and the cells are reduced in number, and in place is found a somewhat homogeneous reticulated substance containing few blood-vessels. These changes are most marked at the ends of the diaphyses. Separation of the epiphyses may occur in severe cases as a result of slight trauma, but is uncommon. The most characteristic lesion, however, consists in the occurrence of hemorrhage depending upon weakness of the blood-vessel walls. This is always present beneath the periosteum, oftenest in the bones of the lower extremities but frequently elsewhere as well (Fig. 210). Hemorrhage may take place, too, in various other tissues of the body. It is liable to be observed in the muscles and skin about the periosteal lesion, or in the neighborhood of the joints, producing a large, tender swelling, easily recognized during life. Small hemorrhages may occur in any of the serous or mucous membranes, often from the kidneys, sometimes from the stomach or intestine, or in the internal organs and bone-marrow. The commonest seat of hemorrhage next to that of the periosteum is the mucous membrane of the gums, especially about the upper incisor teeth. Considerable attention has been directed

¹ Die Vitamine, 1914.

² Zeit. f. Heilk., 1912, LXXII, 1.

³ Virchow's Archiv, 1912, CCVIII, 367.

⁴ Frankfurter Zeit. f. Patholog., 1913, XIV, 1.

⁵ Zeit. f. Heilk., 1912, LXXII, 121.

⁶ Bost. Med. and Surg. Journ., 1913, CLXIX, 232.

⁷ Beiträge z. path. Anat. u. z. allg. Path., 1901, XXX, 232.

to the "white line" first described by Fränkel¹ existing in x-ray plates at the junction of the epiphyses and diaphyses in the long bones in cases of scurvy. In the print the line of course is black. It would appear to be a reliable diagnostic symptom (Figs. 211 and 212).

Symptoms.—Although anemia, loss of appetite, and irritability usually precede the more characteristic symptoms, the first manifestation observed in most cases is *pain in the limbs*. This was reported present in 314 of the 379 cases of the American Pediatric Society's series, in all but 3 of these being in the lower extremities, either alone (149 cases) or combined with pain elsewhere. It often develops so suddenly in infants

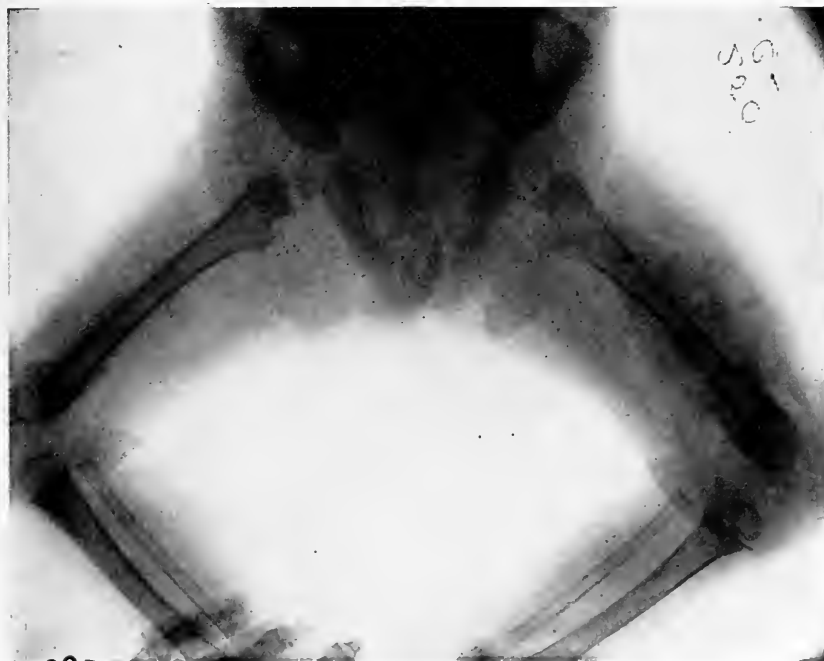


FIG. 210.—RADIOGRAPH OF SUBPERIOSTEAL LESIONS IN INFANTILE SCURVY.

Child of 11 months, admitted to the Children's Hospital of Philadelphia, Jan. 25. Well until 3½ months previously, when pain and tenderness without swelling developed in the left thigh. A month later the right side became tender and swollen, and has continued so. Condition had been supposed to be sarcoma. Radiograph (viewed postero-anteriorly) shows decided swelling about the right femur, and beginning involvement of the left. Orange-juice given freely. Discharged, entirely well.

who have appeared perfectly well, that it may be attributed by the parents to some accident. In other cases the onset is slower, the pain at first being intermittent, and the child often passing a number of days at a time in entire comfort. But in all severe cases the pain soon becomes constant, the tenderness intense, and the slightest attempt at passive movement occasions loud outcries. When the localization can be determined, it is found oftenest in the neighborhood of the ankles or knees. Even approach to the bed-side may cause the infant to scream through fear of being touched. In the majority of cases there is suffering only when the limbs are moved. In 91 of the American Pediatric Society's series, how-

¹ Fortschr. aus d. Gebiete der Röntgenstrahlen, 1906, X, 1.

ever, pain was reported present even when the infant was entirely at rest. In nearly all instances some degree of *pseudoparalysis* exists, dependent upon pain, and in some cases the legs may be kept so still that polio-myelitis or other form of paralysis is suspected. The absence of motion may range from mere disinclination to move the limbs up to complete disability; varying with the case. At times this is the first symptom



FIG. 211.—INFANTILE SCURVY. WHITE LINE.

Subacute case in an infant admitted to the Children's Hospital of Philadelphia, aged 10 months. Knees swollen; tibiae tender on pressure; moves the legs very little; gums swollen and mottled. Radiograph taken Jan. 17 shows the "white line," (in the negative; naturally black in the print.)

noticed (36 of the American Pediatric Society's series). Thoracic pain is sometimes produced by lifting the child in the ordinary manner.

About the same time with the development of pain the characteristic *affection of the gums* develops. It was seen in 313 cases in the American Pediatric Society's series and was the first symptom in 42 cases. It consists, when well developed, in a deep bluish-purple, spongy swelling



FIG. 213.—INFANTILE SCURVY.

Infant of 8 months. Well nourished. Fed upon Horlick's Malted Milk. Fretful, nervous, pain in the legs. Had been suffering for 3 weeks. Recovered in 3 days. Illustration shows the swollen, purple gums about the upper incisors, and the purple streaks in the gums of the lower jaw.

of the mucous membrane, generally over the upper incisor teeth, sometimes so decided that the teeth become entirely concealed (Fig. 213). Slight hemorrhage from the gums, or from the palate or pharynx takes place readily and ulceration is not infrequently present. As a rule the involvement of the gums occurs only in infants whose incisor teeth have already erupted, but this is because the disease is seen so much oftener in



FIG. 212.—INFANTILE SCURVY. WHITE LINE.

Same case as in Fig. 211. Radiograph made Feb. 11. Treatment with orange-juice was instituted, and symptoms were promptly relieved. Radiograph shows the disappearance of the "white line"

those past the age of the first appearance of teeth. In 45 cases in the American Pediatric Society's series no teeth had been cut and in 24 of these the gums were affected. Often at this time *swelling along the shaft of the long bones* can be found. This is hard and tender and is situated usually in proximity to a joint, oftenest the ankle or the knee, but does not involve it. The swelling may be bilateral, with the skin over it shining

but usually not reddened (Fig. 214). It may be only slight, but is not infrequently extensive, increasing greatly the general size of the limb. In severe cases the affected limb may become extensively edematous. Quite often *hemorrhages* in the form of ecchymoses or petechiæ are seen elsewhere in various regions of the cutaneous surface (182 of 353 cases in the American Pediatric Society's series). They may be widespread and may be found also on the mucous membrane of the mouth and pharynx; sometimes upon the conjunctivæ. They are often among the earliest evidences of the disease. Orbital hemorrhage occurs in a rather small proportion of cases (49 of the American Pediatric Society's series) producing purplish swelling of the eyelids and protrusion of the eyeball. In severe cases blood may be vomited or passed by the bowel. Hemorrhage from the kidney, usually slight, is commoner than usually supposed, its frequency not depending upon the severity of the case. I have known it to be the only positively characteristic

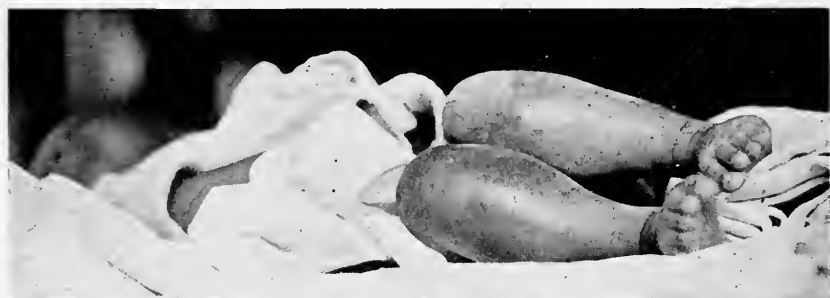


FIG. 214.—INFANTILE SCURVY, SHOWING SWELLING OF LEGS.

Infant of 1½ months, in the Children's Ward of the University Hospital, Philadelphia. Been fed on proprietary food. Pain and swelling in both legs and about the knees. Right arm also involved. Improved on orange-juice, but died of diarrheal disorder.

symptom in otherwise doubtful cases,¹ and have observed it present in probably the majority of cases where the urine has been examined. Blood-casts and granular casts may occur. The loss of blood may sometimes be discoverable only by microscopical examination; in other instances the urine is distinctly red or smoky. Albuminuria independent of the loss of blood is not infrequent.

In the meantime the *general condition* of the child suffers. In severe cases there is a progressive wasting accompanied by the development of a cachectic appearance and one expressing the constant presence of pain. The infant lies almost motionless and may cry almost continuously and sleep but little, and its condition is most wretched. There may be moderate fever. The heart is weak, the appetite diminished, and the stools often diarrheal or constipated. Anemia is little marked at first, but becomes decided as the disease advances, being a secondary anemia with especial reduction of the hemoglobin as compared with that of the red cells and with a moderate and inconstant leucocytosis. Hess and Fish² found the coagulability of the blood slightly diminished, but not to any noteworthy degree.

Complications.—By far the most frequent is rickets, and the association of this with scurvy is so common that a necessary connection

¹ Phila. Med. Journ., 1901, Feb. 2.

² Amer. Journ. Dis. Child, 1914, VIII, 386.

has been supposed. This, however, does not appear to exist, and the two diseases are entirely independent. In 45 per cent. of 340 cases of scurvy in the American Pediatric Society's series it was distinctly stated that rickets was not present, and even admitting that slight rickets may have been overlooked in some of these, it is certainly improbable that it was so in all. Bronchopneumonia or gastroenteric disturbances are occasionally fatal complications.

Course and Prognosis.—The prognosis is excellent in cases promptly treated, and recovery is very rapid. All pain ceases in a week or less. There is scarcely another disease in which the results of treatment are so rapid and remarkable. The swelling of the bones and the constitutional symptoms disappear within 2 or 3 weeks even in severe cases, unless some serious complication be present. If untreated, however, the disease is chronic, the symptoms gradually becoming more marked and the general condition constantly worse and, although spontaneous recovery may eventually take place in the mild cases, death is liable to occur in the severer ones from malnutrition, exhaustion, or some intercurrent disease after a period of several months.

Diagnosis.—Although the disease is usually readily recognized by those familiar with it, it is the source to others of numerous errors. The failure to make a diagnosis nearly always depends not upon any difficulty attending it, but upon the fact that the possibility of the existence of scurvy has not occurred to the observer. The diagnosis in typical cases rests upon the combination of great pain and tenderness of the limbs, with swelling and hemorrhage beneath the periosteum and in the gums and elsewhere. In general it is safe to assume that pain in the limbs and pseudoparalysis developing rapidly in an infant between 4 months and 2 years of age is due to scurvy, unless examination reveals other causes, even although no affection of the gums is discoverable.

The diagnosis of *rheumatism* is the one oftenest falsely made. This disease, however, is exceedingly rare in infancy. When present it affects the joint and not the bone; there is no involvement of the gums, and the employment of anti-scorbutic treatment is inefficacious. The pseudoparalysis of *syphilis* may suggest scurvy. It occurs, however, usually at an earlier age, produces less tenderness, is oftener limited to the arms, is always situated at the epiphyseal junction and not in the shaft of the bone, and is associated with other symptoms of syphilis. When epiphyseal separation occurs in scurvy, the other symptoms of the disease are so well marked that mistakes in diagnosis can hardly occur. *Poliomyelitis* is sometimes suggested by scurvy on account of the apparently flaccid paralysis of the legs. There is, however, in scurvy far more tenderness present and no alteration of the electrical reactions. *Osteomyelitis* invades the joints, if it does not begin there, and is attended by fever and pyemic constitutional symptoms quite different from those of scurvy. The painful immobility of the limbs of scurvy may suggest *hip-joint disease* or sometimes disease of the spine, or the swelling may be mistaken for that of a *malignant growth* of the bone. The frequent presence of rachitis as a complication leads sometimes to the overlooking of the existence of scurvy also. There is, however, no hemorrhagic tendency in rickets, and improvement under dietetic treatment is slow. The hemorrhagic rickets of earlier writers is in reality scorbutus.

Treatment.—**Prophylaxis** consists in the avoidance of diet known to predispose to the development of the disease. Inasmuch as there seems reason to believe that the heating of milk, and especially the employment

of proprietary foods, predisposes, the administration of fruit-juices should begin early. The pasteurization of the food is generally so important a matter that it should never be avoided merely on account of the possibility of scurvy developing. The evils which threaten to follow the ingestion of raw milk not of the very best quality are far greater and much more dangerous than those produced by scorbutus, and the latter are readily prevented.

Treatment of the Attack.—This consists primarily in correcting the diet. In some cases the mere change from a cooked milk-mixture to one of raw milk is sufficient. In others the abandoning of a proprietary food answers. When scurvy appears the change in diet should always be made promptly unless there is some reason against this. On various grounds, however, it may seem inadvisable to make any sudden alteration in a diet which has, in other respects, been found satisfactory. Fortunately we have almost a specific for the disease in fresh fruit-juice, and this should always be administered. Orange-juice is one of the best, the juice of one-half and later of one orange of average size, sweetened, being given either in divided portions or in a single dose, usually between feedings. Diarrhea, if present, offers no contra-indication, and may even be controlled by the treatment. The only fatal case I have seen was one in which orange juice was mistakenly avoided on account of the presence of intestinal disturbance. Freshly expressed beef-juice is also of service, but not to so great a degree. For children over 1 year, or even somewhat younger, potato or other fresh vegetables may be given with advantage. If the diet is for any reason not altered immediately, the change should certainly be made as soon as possible in order to prevent a recurrence of the disease. Should the anemia and debility not disappear promptly iron or cod-liver oil may be needed later. Of course the infant must be handled with the greatest care while the tenderness remains.

CHAPTER III

INFANTILE ATROPHY

(Marasmus. Athrepsia)

This title, probably first employed by Sorano in the 16th century (Albarel)¹ denotes a progressive and finally extreme wasting of the infant without other symptoms or discoverable anatomical lesions to account for it. The term "athrepsia" was applied to it by Parrot.² From this *primary atrophy* must be distinguished all cases of *secondary atrophy* the result of malnutrition dependent upon starvation, congenital syphilis, tuberculosis, or evident chronic disorders of the gastro-enteric tract. This leaves the term infantile atrophy certainly useful, since the condition is common, but unsatisfactory, since many cases so designated are undoubtedly due to positive but unrecognized anatomical causes. The title to a large extent covers the condition designated "decomposition" by Finkelstein (see p. 698), applied to those cases in which the lack of power of digestion and assimilation finally reaches a point at which the infant fails to gain no matter what the diet may be. Whatever the cause of infantile

¹ Ann. de méd. et de chir. inf., 105, IX, 1.

² Prog. méd., 1874, II, 637.

atrophy may be, it is evident that the condition, like icterus, is a symptom rather than a disease.

Etiology.—The disorder is seen chiefly in infants under 1 year living under bad hygienic conditions in cities and fed artificially. It is the cause of many deaths in asylums for infants and in crowded hospital wards, where the title "hospitalism" has often been well applied to it. Some gastroenteric affection frequently precedes it. It has been claimed to depend upon an infection of unknown nature, but of this there is no proof offered. Another theory (Baginsky),¹ assumed an anatomical basis, the lesions being a destructive change in Lieberkühn's follicles, especially in Paneth's cells (Bloch).² The presence of any anatomical lesions has, however, not been confirmed by most investigators. That the disease is not dependent upon the absence in the artificial food of ferments natural to human milk is indicated by the frequency with which infants thrive who are fed entirely artificially. The prevailing view makes primary infantile atrophy a defect in the physiological processes of the organism, as a result of which the infant cannot utilize the food given; this depending either upon disturbance of the intestinal secretions, including ferments, or upon faulty metabolism causing a loss of power of assimilation in the tissues themselves. According to the former view, the intestinal ferment is absent which completes the breaking down of foreign (cow) protein and the reconstruction of the homologous protein which the infant can properly utilize. In the latter theory may perhaps be included the suggestion that an acidosis or some other intoxication develops and acts as a determining cause through the poisoning of the system. It has also been maintained that the imperfect metabolism depends upon defective internal secretions. The presence of diminution in the size of the thymus gland, for instance, has been pointed out by several observers (Mettenheimer)³ (Stokes, Ruhrähnd Royal)⁴ as accompanying atrophic conditions in infancy. No etiological relationship, however, has been proven. Whatever the influence of any of the causes mentioned may be, they are in any event not the primary or sole factor. In the large majority of instances there has early been some long-continued defect in the diet, which has finally resulted in an inability on the part of the infant longer to utilize the food given to it. Very defective hygiene is another influential factor, especially confinement in illy-ventilated rooms in the poorer parts of the cities, or the occupying of crowded institutions for infants. It is on this account that the disease is comparatively uncommon in country practice, or in civic practice among the better classes. A further cause often of importance is the presence of a constitutional debility, whether due to prematurity or to ill-health of the parents.

Pathological Anatomy.—Any lesions found must be regarded only as secondary. Fatty changes in the liver are often seen, but no more frequently than in many other disorders in infancy. Bronchitis, bronchopneumonia, atelectasis, hypostatic pulmonary congestion, and intestinal derangements are also often observed, and may, as complications, have precipitated the fatal ending. The lymphatic glands are sometimes enlarged. Atrophy of the mucous membrane of the intestine may be present but appears to be the result of the general atrophy of the soft tissues of the body, and is frequently entirely absent.

¹ Brit. Med. Journ., 1899, I, 1084.

² Jahrb. f. Kinderh., 1906, LXIII, 424.

³ Jahrb. f. Kinderh., 1898, XLVI, 55.

⁴ Amer. Journ. Med. Sci., 1902, Nov.

Symptoms.—The essential symptom is progressive loss of weight. The emaciation finally becomes excessive, the face wrinkling all over with



FIG. 215.—INFANTILE ATROPHY.

From a patient, aged 7 weeks, in the Children's Hospital of Philadelphia. Had been boarded out since birth. Temperature 95° to 98°F. (35° to 36.7°C.). 2 to 3 soft stools daily. Death.



FIG. 216.—INFANTILE ATROPHY.

Child of 4 months, admitted to the Children's Hospital of Philadelphia, weighing 6 pounds, 4 ounces (2835). Been fed on condensed milk and various fresh-milk mixtures. Progressive loss of weight. No active digestive disturbance until within 2 weeks.



FIG. 217.—INFANTILE ATROPHY.

Same infant as in Fig. 216, taken 2 months later, weighing 10 pounds (4536), after careful dietetic and other treatment.

each feeble cry, giving the infant the expression of a withered old man (Fig. 215, 216 and 217). The fontanelle is depressed and unusually small;

the bones of the skull overlap; the chin and cheek-bones are prominent; the eyes large and sunken; the skin of the body is pale and hangs in loose wrinkled folds. The arms and legs seem to consist of bones with only a thin layer of skin over them; the hands are like claws and they and the feet cold and cyanosed. The outlines of the collar bones and the ribs suggest a washing-board. The abdomen is sometimes very prominent; sometimes sunken with the thin skin over it showing dilated veins and revealing the outlines of the intestines beneath. The temperature is usually subnormal (Fig. 218), the respiration superficial, the circulation poor, more or less anemia is present, and, toward the end of life, edema also, especially of the face and extremities, but sometimes of the whole surface. At this period a gain in weight may give false encouragement. It is, however, a bad symptom, due to the deposit of liquid in the tissues. The appetite is usually diminished, sometimes voracious; the stools are often regular and well digested, oftener contain mucus and undigested food. Yet no matter how normal the stools, the loss of weight continues. Vomiting occurs easily; albuminuria is absent. The infant is at first fretful and cries often; later apathetic and lying with little movement. The muscles are usually flabby and relaxed, but in some cases a condition of hypertonia is observed, producing arching of the back, retraction of the head, and flexion of the thighs upon the abdomen. (See Vol. II, p. 254.) The whole aspect is one of the most shocking in the realm of pediatrics. The infant appears to be merely a skeleton with a thin covering of skin.

Complications.—Infantile atrophy is often complicated by the development of furunculosis and other cutaneous abscesses; erythema chiefly of the nates, scrotum, and back of the thighs; hernia; and thrush. Petechiae, especially on the abdomen, often develop shortly before death. (See Vol. II, p. 477, Fig. 399). Bronchopneumonia, atelectasis, and gastrointestinal derangements are frequent. Convulsions may terminate the scene.

Prognosis.—This is always grave and the majority of well-established cases die after weeks or months of wasting, in spite of the greatest care. The disease steadily grows worse, or in some cases exhibits temporary periods of arrest of loss of weight or even a slow gain, to be followed by

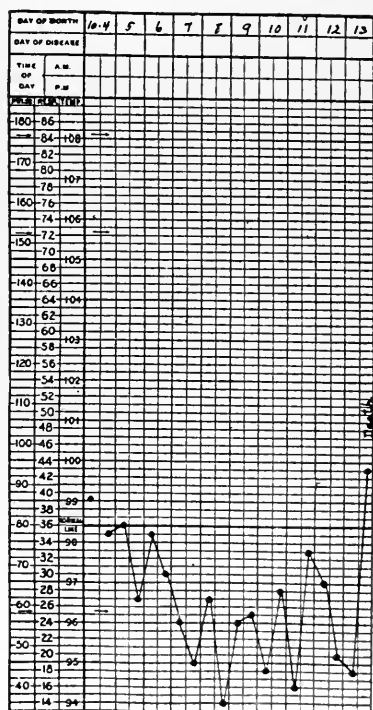


FIG. 218.—INFANTILE ATROPHY, WITH HYPOTHERMIA AND TERMINAL INCREASE OF TEMPERATURE.

Robert M., aged 3 months. Said to have been vomiting more or less since age of 3 weeks. Bottle fed. Last 2 weeks some retraction of head. Entered in the University Hospital in Philadelphia, Oct. 4, cries a great deal, much emaciated, no vomiting since admission, stools normal. No improvement. Death. Autopsy findings negative.

sudden, rapid renewed loss. The reduction of the amount of food taken, on the ground that some digestive disturbance is present, is followed by increased loss of weight; and the same happens if the quantity is augmented. In fact, marantic infants are extremely susceptible to changes in the nourishment, and may be made rapidly worse by this. Death may occur quite unexpectedly from causes not ascertainable, the infant being perhaps found dead in bed, although when last seen it was apparently no worse than usual. In other cases some complication, often of very slight severity, is the cause of rapid failure of strength and of a fatal ending. The sudden development of hot weather precipitates the termination. Yet under favorable circumstances a certain number of cases can be saved. The longer the condition has lasted and the older the infant, the greater the prospect of cure. Recovery may be slow but finally complete (Fig. 217), and the child in later years may appear no worse for the illness of infancy.

Diagnosis.—The condition does not differ in symptomatology from the secondary atrophy dependent upon starvation, tuberculosis, or other cause, and the object of diagnosis is to exclude these factors. This is at times most difficult. The existence of tuberculosis may become evident only at autopsy. In other cases the presence of the tuberculin reaction or the discovery of some localized symptoms indicate tuberculosis. A coincidence of the date of the commencement of failing health with that of weaning or some other change of diet suggests marasmus. Starvation from mere lack of sufficiently nourishing food is readily differentiated by the rapid improvement which follows a proper diet.

Treatment.—In the way of prophylaxis the continuance of breastfeeding in whole or in part, or, when this is not possible, the careful avoidance of digestive disturbances, accomplished by a judicious selection of cow's milk mixtures is of great value, as is the obtaining of an abundance of fresh air and the avoidance of excessive summer heat. For the developed condition, dietetic measures are of much service, yet these can be but experimental as long as the nature of the cause is not understood. The employment of a suitable wet-nurse is greatly to be desired, in fact is almost imperative, although it by no means follows that life can be saved in this way. When it is impossible to feed otherwise than artificially, the same rules hold good, and the same difficulties obtain, as attend the treatment of indigestion. (See p. 767.) Care must be taken in advanced cases to avoid any prolonged starvation-treatment, such as would be properly employed in acute digestive disturbances. The strength of the infant is not sufficient to tolerate this well. On the other hand, the initial food should be of only such a caloric strength as would, under normal conditions, maintain the weight, without effort to increase it. More than this cannot be hoped for at the beginning. The food should be weak, given frequently and in small amounts. As regards the selection of the nourishment, the principles apply which are operative in the treatment of chronic digestive diseases (pp. 723, 763). In general protein-foods such as buttermilk and casein milk offer the best of the unfortunately often slim chances for success. Dextrin-maltose preparations and saccharose are usually preferable to lactose. Weak cereal decoctions may sometimes be of benefit. Fat is generally better avoided or given in very small amount; although it sometimes happens that cream-whey mixtures are certainly of value.

In cases where wasting is great and the need of liquid in the tissues evident, hypodermoclysis may often be used with advantage. Freeman¹

¹ Arch. of Ped., 1917, XXXIV, 428.

claimed good results from the subcutaneous injection of horse-serum, and Dunn,¹ from the introduction of a 5 per cent. solution of dextrose into the longitudinal sinus; giving of the solution $\frac{1}{60}$ of the body-weight. This supplies the infant with much needed nourishment as well as with liquid.

Of great importance is the obtaining of fresh air. The disease diminishes in hospital practice in proportion as the air-space of the room is increased, and especially through the use of open sun-parlors or roof-gardens, unless the presence of unusually low body-temperature precludes this. In fact in hospital-practice one of the best plans of treatment is to send the child with beginning hospitalism home, if it can be taken care of there in a manner at all suitable. The fact, however, is too often evident that the bad hygienic conditions of the home are even worse than the retention of the infant in the hospital.

General massage with oil is of value in many instances. Alcoholic stimulants in moderate dose are serviceable if the fontanelle is much depressed and the general circulation poor. Should the temperature of the body be below normal it must be maintained by the employment of hot water-bottles. In the case of young infants with threateningly low temperature it is best to confine the patient to a suitable warm room, which must, however, receive sufficient previously warmed fresh air.

CHAPTER IV

MALNUTRITION

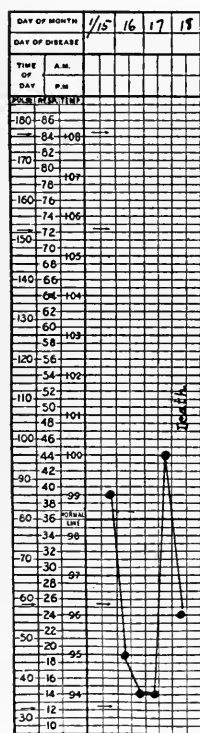
By the general and rather vague term Malnutrition we may cover all the secondary atrophies and instances of failure to gain in weight, independent of whether the causes are organic or functional; reserving the title "Infantile Atrophy" or "Marasmus" for the severe cases with the etiology and symptomatology as described in the previous chapter. The term Inanition, literally "emptiness," may be regarded as expressing an unusual degree of malnutrition, in reality dependent upon a starvation. The distinction is, however, usually not very sharply drawn.

Etiology.—Malnutrition is extremely common, and depends upon various causes. A constitutional debility may be present in children from the time of birth, and render them always below normal in their strength, weight, and resisting power to disease of any sort. Many cases of premature birth are to be placed in this category. Children with a congenital, excessively nervous development constitute a considerable proportion of the instances of malnutrition in childhood. Not only may tuberculosis and syphilis of the parents produce a state of malnutrition in the offspring, which, however, may not show evidences of the existence of either of these diseases, but parents delicate or diseased from any cause may be unfit to produce children capable of thriving normally.

In probably the majority of cases, however, the condition is not a constitutional one observable at birth, but is acquired later, the infants appearing healthy when born. The presence of chronic disease of any nature may be the cause, to be mentioned here being tuberculosis, syphilis, rachitis, diabetes, malignant growths and chronic disturbances of any of the organs of the body, most frequently the gastroenteric tract. Even the occurrence during early infancy of an acute disorder, oftenest digestive in nature, may leave a chronic malnutrition in its train. Continued

¹ Arch. of Ped., 1917, XXXIV, 425.

improper or insufficient diet and the constant living under bad hygienic conditions with lack of sufficient fresh air are perhaps the most frequent factors; and this applies not only to the children of the poor to whom too



(Czerny and Keller).¹ In the United States it is certainly not so common. Sometimes it follows the too long continuance of an amylaceous diet, prescribed for infants who have been suffering from diarrhea with inability to digest milk. It is to be noted that the occurrence of this form of malnutrition generally develops only when such a diet has been continued for a considerable time. The temporary substitution of milk by starchy food is a well-approved course of treatment of some digestive disturbances; but the danger of the continuance must be borne in mind. The appearance of symptoms would appear to depend not so much upon the presence of the excess of starch as upon the relative deficiency of protein and of fat, the disease being one of deprivation.

Three types may be recognized: First the hypertonic form (see Vol. II, p. 254) in which there is a rigid condition of the muscular system with opisthotonos, adduction of the arms and thighs, and flexion of the forearms and legs. This is the least common variety; and hypertonia may be seen in other nutritional disturbances than those dependent upon a starchy diet. Second, an atrophic form which does not differ much in appearance from ordinary inanition from starvation, although the condition of hypertonia is often associated. In this variety there has been no addition of salt to the diet. Third, the hydremic form in which salt has been added freely to the food and as a result the water is retained in the tissues. This retention of water makes the infant at first appear well nourished, but gradually the presence of edema becomes very noticeable, and the increase in weight is decided. In all forms the skin is pale and of a pasty appearance, and the infant is especially liable to the development of infections, chief among them being suppurative processes of the skin and xerosis of the cornea and conjunctiva with destruction of the eye often following.

In older children the symptoms of malnutrition show themselves in various ways, such as anemia; coldness of the extremities; the weight and often the length being decidedly below normal; fretfulness; nervousness; poor appetite; constipation; coated tongue; insomnia; fatigue on slight exertion, the child being unwilling to exercise, or too restless and active and growing fatigued and irritable before evening comes on. Sometimes the children are above the average mentality; sometimes they easily become mentally tired, and the ordinary school-work is difficult and burdensome. Very slight causes are liable to produce outbreaks of indigestion. There is very little resisting power to attacks of disease of any sort, illnesses developing readily and leaving a distinct impression on the system.

Course and Prognosis.—This varies with the age and with the cause. Children who are born constitutionally predisposed to malnutrition cannot be expected to become hardy with any rapidity if at all. In those in whom the disorder depends upon some existing or previously existing disease, the duration of the condition and the ultimate outcome depend upon the nature of this and the possibility of removing it if still present. Very often the state of malnutrition disappears to a large extent when later childhood is passed; in other cases it is permanent. There is always, too, the danger of a fatal termination from the occurrence of some of the numerous complicating disorders to the development of which the subjects of malnutrition are especially liable. The immediate danger from malnutrition is much the greatest in young infants. The older they grow the greater the chance of final recovery, provided proper therapeutic measures can be enforced.

¹ *Des Kindes Ernährung*, etc., 1906, II, 62.

In the nutritional disturbances dependent upon an excessively starchy diet, the prognosis is very uncertain, being worse in proportion to the youthfulness of the infant, the length of time the faulty diet has been employed, and the duration of the symptoms. The mortality in young infants is high. Yet patients will sometimes improve rapidly when a proper diet is instituted. The danger from infections of different sorts is great, and the infants appear to have but little resisting power to them.

The acute inanition from insufficient nourishment generally offers a favorable prognosis, if it has not advanced too far; and recovery is rapid when the dietetic defect is remedied. Very often, however, the patient has developed through lack of food an intolerance for it, and the prognosis is then unfavorable. In this connection must be emphasized the danger of starvation-treatment as the first step in the course of the management of cases of indigestion in infants much debilitated. It is the proper treatment in acute cases with good general health; but in those with but feeble strength it may precipitate a most dangerous state of inanition if continued unduly.

Treatment.—The treatment of malnutrition depends naturally upon the cause. Inasmuch as this is so often dietetic and hygienic, the first effort must be made to discover what may be the error in this particular and to correct it.

As regards the malnutrition of *infants*, inanition occurring in those breast-fed requires a careful study of the composition and amount of milk secreted, as well as the determining, in the event that nothing is wrong in this particular, whether the infant, perhaps weakly, is actually drawing sufficient nourishment from the breast. It may be necessary to pump or express the milk and to feed it from a bottle or dropper or by gavage. In infants artificially fed who refuse their nourishment, feeding by gavage may be necessary. In young infants in whom the state of malnutrition depends upon digestive disorders, the securing of breast-milk is often an essential for recovery. This may be fed in small amounts at frequent intervals, or in larger quantities with longer pauses, depending upon the attendant symptoms and the results obtained. When the employment of substitute-feeding is unavoidable, the food may be carefully selected according to the indications of the symptoms. (See Chronic Gastritis, p. 723; Chronic Intestinal Indigestion, p. 763, etc.) In general the high-protein foods, such as casein-milk and buttermilk, are most efficacious. Sometimes malt-soup gives excellent results; in other cases whey, white of egg, and the like, are valuable as temporary expedients. It is necessary to make sure that the food is of sufficient strength to supply the caloric needs. In the case of malnutrition dependent upon excessive amount of starch, the dietetic remedy is obvious. Inasmuch as the trouble is not so much the starch as the nearly or complete absence of fat and protein and possibly of salts, these ingredients must be supplied and the starch withdrawn or reduced in amount.

Next to the management of the diet in infancy that of the hygiene is of the most importance. The chief error here, both among poor and rich, is deficient fresh air, the child being confined by night and often by day to close, poorly ventilated rooms. Whatever risk there may be from exposure, that of keeping the infant housed is certainly much greater. There is naturally a middle course advisable in all cases of much debilitated infants, and enthusiasm for open-air treatment should not lead to exposure to chilling, especially in patients with a tendency to low body-temperature, who bear this very badly. The employment of hot water

bottles and the careful screening against cold wind will permit of open-air treatment in many cases, while in others the keeping of the infant in a well-warmed room with the windows open may constitute a satisfactory compromise.

A number of other measures may be serviceable, according to the needs of the case. In atrophic infants with vomiting or diarrhea, who are taking or retaining but little nourishment, frequently the most pressing need is that of water in the tissues. In such an event enterocolysis with normal saline solution is often of the greatest value. When it is not satisfactorily retained, hypodermoclysis may be required. Cardiac stimulants should also be used as the occasion demands. In other cases of malnutrition gentle massage, such as the mother herself can give, is of service.

In the treatment of malnutrition in *older children* the causes are so manifold that only a careful study of the conditions which have obtained from infancy onward can guide to a successful result. Here, too, the diet is often at fault, although not so predominately as during infancy. In all classes of society the greatest ignorance and carelessness is often shown by parents in these matters. The appetite for the meals is spoiled by allowing cakes, candies, and the like between meal-times. The fancies of the child are made the guide rather than what is best for it; sometimes on the ground that if denied the manifestly improper articles of food it will take nothing. The ultimate result is, however, always bad. The diet should be simple, yet sufficiently varied if food of one nature palls, as is often the case. Whether the child shall be urged to eat depends upon the individual. As a rule, if voluntarily going without a meal, the patient will often have a better appetite for the next and will make up the deficiency. Yet some children seem to have been born with a constitutional lack of appetite, and in these unobtrusive persuading to eat is often a necessity. This is, however, frequently much overdone by parents, and awakens in the child a hysterical objection to the taking of food of any sort. (See "Anorexia nervosa," p. 706.) When the appetite is poor proper selection should be made of the sort of food which will nourish most. Thus, for instance, I have seen children satisfy themselves when beginning a meal with a thin soup, and refuse the following more substantial substances; and have succeeded in modifying this by giving the soup as a dessert, it making no practical difference whether this article was then declined or not.

The whole course of the child's daily life must be supervised by the physician. There is in many instances far too much confinement to the house or in school, and far too much pushing in an educational line. The child spends the greater part of both morning and afternoon in school, and on returning home has lessons in piano-music, modelling, foreign languages, drawing, and so on. There is no opportunity given for active outdoor amusement, and it is not long until a distaste for this arises, and the patient has to be driven from the house to partake in it. In other children, on the other hand, the fondness for outdoor sports exists to such an extent that long before evening the child is in an over-tired state, ready to cry on the slightest provocation, and a night troubled by insomnia follows. Parents often fail to remember that a child never knows enough to stop play before becoming tired out. The same nervous condition arises from over-excitement of any other sort, such as going frequently to the theater, moving-picture shows, children's parties, and the like.

Enough has been said to illustrate the great necessity of a thorough study of the child's daily life and the proper regulation of this. Certain other therapeutic measures may be considered. Massage and gymnastic training are useful for children who take too little exercise, but not suitable for those who take too much. Rest, recumbent, in the middle of the day, not insisting upon sleeping in the case of those in later childhood, is sometimes remarkably efficacious. A cool morning sponging or affusion is often an excellent stimulating measure if the brisk after-rubbing brings about a good reaction. The bath-room should be warm and the child should stand in the tub in a few inches of warm water, this being used for cleansing purposes. Finally, the common observation that the children enjoy the best health when out of town during the summer months, indicates that in many instances life in the country during the whole year is what will do most good.

The employment of drugs occupies a very minor position in all cases of malnutrition of any age. They should be used chiefly symptomatically as the need arises. Particular attention must be paid to the relief of the chronic constipation which is so often an attendant. The anemia which is a frequent symptom may need a prolonged course of iron. Cod-liver oil is excellent in many instances, provided it does not produce coating of the tongue and loss of appetite. Children usually take it readily if made into an emulsion or given combined with a syrupy extract of malt.

CHAPTER V

RHEUMATISM

Rheumatism is often classified among the infectious disorders, and certainly shows a close similarity at times to infections of a septic nature. For various reasons, however, it seems more convenient to consider it provisionally among the general and nutritional diseases; at least until such time as the invariable causative relationship of a specific germ may be definitely proven beyond possibility of question.

Etiology. Predisposing Causes.—*Age* is important among these. In infancy rheumatism of any form is very rare. I have seen but 1 instance of articular involvement at this period, occurring in a male infant of 8 months,¹ and Miller writing in 1899² found recorded under 1 year of age only 19 cases, in addition to 1 of his own, which seemed properly to belong to this class. Yet a few instances have been reported which appear to have been congenital (Abraham).³ It is uncommon in early childhood, but after this period, especially in some of its varied forms, steadily increases in frequency. Langmead⁴ analyzed the conditions existing in 2556 school children from 3½ to 14 years of age, and found definite rheumatism in 133. The individual predisposition at this time is, however, not very great, Baginsky⁵ finding articular rheumatism in but 1.4 per cent. of 10,375 children coming to the hospital, and Wachenheim⁶ 113 instances in about 8000 children. Girls appear to be attacked rather oftener than boys. In the fatal cases in children there are about 3

¹ Arch. of Ped., 1908, April.

² Arch. of Ped., 1899, Sept.

³ Med. Rec., 1896, L, 547.

⁴ Lancet, 1911, II, 1133.

⁵ Berl. klin. Wochenschr., 1904, XLI, 1213.

⁶ Arch. of Pediat., 1908, XXV, 669.

females to 2 males (Poynton, Agassiz and Taylor).¹ Inheritance is a factor of importance, certain families seeming especially predisposed, either the parents or other children having suffered from the disease. Kephallinos² discovered evidences of inheritance in 50.7 per cent. of 69 cases. Defective hygiene, especially exposure to cold and dampness or to sudden alterations of temperature, is perhaps the most influential predisposing factor. For this reason rheumatism is more prevalent in the cooler and damper season of the year.; and this, too, explains the fact that some years and some countries produce many more cases of rheumatism than others.

Exciting Cause.—The direct cause of the disease is unknown. Excessive acidity of the blood, dependent especially upon lactic acid, has been claimed to be influential, but there appears to be no proof of this. Microorganisms have been discovered in the blood, heart and joints by various observers, probably first by Popoff in 1887.³ One of the most important contributions is that of Poynton and Paine,⁴ who found cocci (*streptococcus rheumaticus*) in fatal cases of rheumatism, present in the joints, blood, heart and tonsils. They considered these the cause, and that they probably entered by way of the tonsils, since rheumatic individuals appear especially predisposed to tonsillar inflammation, and the characteristic rheumatic symptoms are in many cases immediately preceded by an acute angina. Later studies by them⁵ confirmed their past experience, and Coombs, Miller and Kettle⁶ and others produced arthritis and carditis in rabbits inoculated with streptococci obtained from rheumatic individuals. Whether or not any variety of microorganism can be considered the specific cause remains to be proven. Certainly, if so, there must be some powerful constitutional susceptibility present as well. Family outbreaks, it is true, point decidedly to an infectious nature of the disease (Allaria)⁷ yet but few such instances have been reported, and it is difficult to understand the association of infection with the sudden development of such nervous manifestations of rheumatism as seen, for instance, in many cases of chorea.

Pathological Anatomy.—There are no characteristic articular lesions in acute cases beyond hyperemia with turbidity of the fluid, and slight infiltration of the neighboring connective tissue. As a rule no bacteria are found, in the experience of most observers. Purulent inflammation occurs only as a complication. In the chronic cases there is decided effusion into the joint, with thickening of the capsule and ligaments, erosion of the cartilage, and finally involvement of the bones resulting in ankylosis. Fibrous nodules are sometimes present beneath the skin in various regions in children. These consist of connective tissue of an inflammatory nature, fibrin, and cells. The lesions of rheumatic endocarditis and pericarditis will be considered in the chapter upon Diseases of the Heart.

Symptoms.—For the correct understanding of the manifestations of rheumatism in children, the idea must be banished that the affection

¹ The Practitioner, 1914, XCIII, 445.

² Wien. klin. Woch., 1900, XIX, 563.

³ Medit. Prebavlonia K. Moskowa Sbonieskie, 1887, 101. Ref., Dunn, Journ. Amer. Med. Assoc., 1907, LXVIII, 493.

⁴ Lancet, 1900, II, 860.

⁵ Lancet, 1910, I, 524.

⁶ Lancet, 1912, II, 1209.

⁷ Revista crit. di med. clin., 1901, Nov. 23. Ref., Brit. Med. Journ., 1902, Jan. 11. Current Lit., 5.

must show itself in the same manner as in adults. The symptoms of rheumatism in early life are manifold. While an acute arthritis is the type, yet there may be no involvement of the joints at all, or this may occur secondarily. Particular notice must be taken of the vague, trifling pains in the joints or muscles, which are ignored by parents, but which can readily be followed by affections of the heart or by chorea. Cardiac involvement is especially common in children and is often the

first manifestation. Tonsillar inflammation or torticollis may be quickly followed by arthritis, or, even without this, by pericarditis or endocarditis. Chorea may succeed articular inflammation or may precede it, or be followed by cardiac inflammation without any arthritis. Subcutaneous fibrous nodules may be the first symptom or may occur subsequently to articular disease. Indeed, any one of these conditions may be the first to appear, but is liable soon to be followed by others. Although, therefore, articular rheumatism may be considered the type, the other conditions are rather to be considered forms of rheumatic manifestations than complications.

Acute Articular Rheumatism.—Occasionally preceded by malaise, or sometimes by sore throat for several days, the attack generally begins acutely with fever, loss of appetite, and swelling, pain, and redness in one or several joints. The intensity of these symptoms varies greatly. In early childhood they are less marked than later, and at any time in early life the articular symptoms are usually not so decided as in adults, the redness, swelling and pain are less evident, sweating absent or slight, and the fever is less, reaching 103° to 104°F. (39.4° to 40°C.) at the onset but soon falling to about 101°F. (38.3°C.) (Fig. 220). Generally the joints of the lower extremities are first affected, the ankle or knee on one side being much oftenest attacked, with the hip usually next in order of frequency. The disease may remain limited to a single joint, but as a rule the corresponding joints

of the other side are soon involved also, and perhaps the wrists, elbows and shoulders as well; or not infrequently the joints of the upper extremity may be attacked alone or primarily. Sometimes the disease appears in the cervical vertebrae, less often in the fingers and toes or other joints. Quite often one joint improves as another becomes affected, but it is not uncommon for the inflammation to reappear in the first joint as the course of the disease progresses. As a rule not many joints are involved.

In the mild attacks, such as are seen especially in early life, the

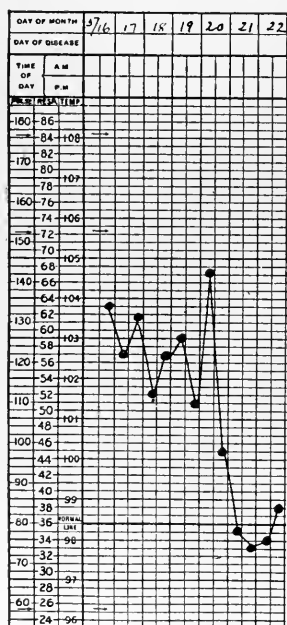


FIG. 220.—ACUTE ARTICULAR RHEUMATISM.

Alice W., aged 6 years. After becoming overheated by skating, developed headache, fever, and pain in the knees, ankles and feet. On entrance to the Children's Ward of the University Hospital, Philadelphia, May 16, exhibited pain, redness and swelling of both knees, tongue coated. No cardiac murmur present. Leucocytes 30,200; May 22, swelling and pain nearly gone. A faint systolic murmur audible.

patient may not feel ill, the fever is insignificant, and the child is not confined to bed unless the lower extremities are affected. Often there is only sufficient pain to produce lameness without actual inability to walk. The discomfort is frequently so insignificant that it is given by the parents the common title of "growing pains."

The well-marked typical attacks of the adult type are not often seen until toward the end of later childhood. Here the expression of the face is one of pain and the tenderness of the inflamed joints is exquisite, any change of position causing severe suffering. The urine is high-colored and diminished in amount and usually acid, and there is abundant acid, sour-smelling perspiration. The blood in these cases exhibits a moderate leucocytosis, and anemia develops if the case is long continued. There



FIG. 221.—CHRONIC ARTICULAR RHEUMATISM.

Showing enlargement of the elbows, hands and knees. Same case as in Fig. 222.

is coating of the tongue and loss of appetite. The temperature remains elevated, sometimes with exacerbations as fresh joints are attacked. Delirium and other cerebral disturbances are rare at any period of childhood.

The *duration* of acute articular rheumatism is variable. In average cases it is from 1 to 2 weeks, but the remarkable tendency to relapse may lengthen the course very greatly. Some cases pass into the chronic form, but this is much less common than in adults.

Chronic Articular Rheumatism.—Under this heading may be included disorders described as rheumatoid arthritis, arthritis deformans, and by other titles. The relationship of these to each other and to acute articular rheumatism is still far from determined. Provisionally they may all be considered as manifestations of chronic rheumatism, inasmuch as it seems impossible to draw any sharp line of demarcation between them. Chronic rheumatism is, fortunately, uncommon in early life. Ibrahim,¹

¹ Zeit. f. orthop. Chirurg., 1914, XXXIV, 213

however, reported upon 273 collected cases including certain of his own. Slightly more than $\frac{1}{2}$ were less than 6 years of age. I have seen a very considerable number of instances. It may follow an acute attack without any cessation of symptoms, or may be the final result of a series of relapses or recrudescences. A second class of cases has certain points of difference, yet cannot be clearly distinguished. In this the course is subacute or chronic and progressive from the beginning (*Rheumatoid arthritis*) (Fig. 221). Either form may begin in the larger joints usually attacked in acute articular rheumatism, but there is a tendency to primary localization in the hips, small joints of the fingers, jaws, or the

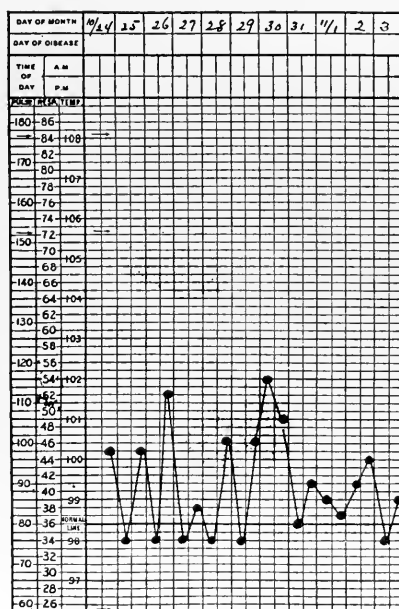


FIG. 222.—CHRONIC ARTICULAR RHEUMATISM.

Ignatio C., aged 7 years. At 20 months developed painful swelling of knees and fingers. In bed 3 or 4 months. Then entirely well until Dec., 1912, when gradually all joints of lower limbs, the hands and the elbows became swollen and painful, neck stiff. Been confined to bed ever since. Admitted to the Children's Hospital of Philadelphia, Oct. 25, 1913. Poorly nourished; very bad teeth; wrists, elbows, ankles, fingers and toes swollen but not tender; neck stiff; fixation at hips; knees swollen and tender; spleen not enlarged. Leucocytes 12,000. Von Pirquet reaction negative. Teeth treated by dentist. Removed unimproved.

neck. This is especially true of the second class of cases. The disease spreads from joint to joint without disappearing in those first affected, until the majority of the articulations are involved. There is increasing debility and anemia, and a tendency to cold, clammy sweating. Endocarditis may occur in either group, but less often in the second. Fever is usually moderate (Fig. 222) or absent. Periods of improvement occur but usually without complete disappearance of symptoms. At these times pain on movement may be moderate, but it becomes severe during the exacerbations. The joints become swollen and distorted and grate when moved; the skin over them shining and the neighboring muscles atrophied. There is involvement of the cartilages and bony structures

and the periarticular tissues, and the final production of great deformity of the joints (*Arthritis deformans*) and of ankylosis. A special form of chronic arthritis has been described by Still¹ which is possibly rheumatic, possibly tuberculous, or dependent upon some chronic infection. It is characterized by progressive involvement of the joints, which become enlarged and stiff but not very painful, but which do not undergo destructive processes. There is more or less fever and always enlargement of the spleen and of the lymph nodes.

There is reason to believe that all the forms of chronic articular rheumatism depend upon a chronic infection arising from the tonsils, sinuses, joints, or the roots of the teeth. It has also been claimed that it may be dietetic in origin (Pemberton).²

Cardiac Rheumatism.—As already stated, this is sometimes the first manifestation of the disease, being evidenced by fever and vague general symptoms, the cause of which is unknown until a cardiac murmur becomes audible, and perhaps other characteristic symptoms developing later prove that the affection is of a rheumatic nature. Oftener, however, cardiac symptoms are secondary to chorea or arthritis. Involvement of the heart is especially liable to occur in childhood, and although more frequent during the severe attacks of articular rheumatism, it is common even in the mildest. In fact, the articular inflammation may have been so slight as to have been overlooked. Endocarditis is the most frequent lesion. A certain degree of this is present in probably the majority of cases of articular rheumatism, as shown by the acceleration of the pulse-rate, which is so often out of proportion to the elevation of temperature. It appears usually about the end of the 1st week of the articular attack, or earlier. Coombs³ found cardiac lesions in 60 per cent. of 75 children with rheumatic disease, and Kephallinos⁴ in about 70 per cent. of 129 cases. A valvular lesion, generally mitral, persists in probably $\frac{1}{2}$ or more of the cases of articular rheumatism in children, developing in later attacks if not in the first. Pericarditis, although less common, is very characteristic of rheumatism in children, occurring in from 10 per cent. to 20 per cent. of the cases of articular rheumatism. It is seen much oftener than in adult life. Nearly always accompanied by endocarditis, it has generally a sudden, acute onset, followed by effusion, and finally often by more or less obliteration of the pericardial sac. (See Diseases of the Heart, Vol. II, pp. 113, 140, 141.)

Chorea.—This is a frequent manifestation of rheumatism, although not all instances of chorea can be called rheumatic. In 226 cases of chorea reported by Still⁵ at least 126 (55.7 per cent.) exhibited rheumatism. The question of the relationship of the two conditions is further discussed in the chapter upon Chorea. It may be the primary disorder and may be followed by arthritis, but oftener, if the combination exists, the latter is the first to develop. As the symptoms of chorea appear, those of articular inflammation may suddenly cease. Very frequently it occurs without any articular involvement whatever, and is then often followed by endocarditis. All three conditions may be present at the same time.

Subcutaneous Fibrous Nodules (Fig. 223).—Although first described by Meynet⁶ this affection has been most frequently recorded by English

¹ Med. Chir. Transac., 1897, LXXX, 47.

² Amer. Jour. Med. Sci., 1914, CXLVII, 423.

³ Bristol Med.-Chir. Journ., 1907, XXV, 193.

⁴ *Loc. cit.*

⁵ Practitioner, 1901, LXVI, 53.

⁶ Lyon méd., 1875, XX, 495.

physicians. Barlow and Warner¹ in 1881 reported upon 37 cases in children. In the United States it certainly appears to be uncommon, and the same would appear to be true for Germany (Berkowitz).² I have encountered it in but few instances. It appears to be more frequent in children than in adults. The nodules vary in size from that of a pin-head to that of a small nut, are not reddened or tender, and are found oftenest upon the back of the head, about the joints, especially the elbows, knees and ankles, along the tendons and the vertebræ and upon the pinna of the ear. They are distributed somewhat symmetrically, vary in number from very few to 50 or more and generally develop after the articular inflammation appears, although I have seen them abundant before this. They usually take some weeks to disappear, and may exceptionally last many months.



FIG. 223.—SUBCUTANEOUS FIBROUS NODULES.

Boy of 4½ years. Chorea, chronic endocarditis, nodules about the wrists and ankles. (Berkowitz, *Arch. f. Kinderhk.*, 1912, LIX, 6.)

Cutaneous Manifestations.—Rheumatic subjects are prone to attacks of erythema of various forms, with which articular inflammation is often associated. Purpura too, is sometimes combined with rheumatic arthritis, with or without erythema. It is far from certain, however, that the majority of cases of arthritis with purpura have any connection with rheumatism. (See Purpura, Vol. II, p. 477.)

Tonsillitis.—As already stated, inflammation of the tonsils or pharynx may be promptly followed by articular inflammation or, even without this, by cardiac disease. There seems to be a special tendency to tonsillitis in evidently rheumatic subjects.

Muscular Rheumatism. Rheumatic Myalgia.—The question whether this condition is actually rheumatic has been much disputed. There appears to be no doubt, however, that at least some of the cases of myalgia are evidences of rheumatic disease. This is true, in children especially, of the acute torticollis which is characterized by sudden onset, great soreness and stiffness of the neck chiefly in the sternocleidomastoid muscles, rapid disappearance, and association with tonsillitis or other rheumatic manifestations. It is to be distinguished from rheumatism of the cervical vertebral articulations. I have seen arthritis and cardiac involvement quickly follow torticollis. (See Torticollis, Vol. II, p. 411.)

Another evidence of muscular rheumatism common in children are the so-called "growing pains" located in the extremities, to which little attention is paid as a rule, but which may be followed by cardiac involve-

¹ Transac. of the Internat. Med. Cong., 1881, IV, 116.

² *Arch. f. Kinderhk.*, 1912, LIX, 2.

ment. Most frequently, however, these growing pains are probably located in the joints. Lumbago, pleurodynia, and other localized myalgias are less common in early life.

Complications.—Other conditions less common than those described may complicate rheumatism, some of them exceptionally. Among these are vomiting, peritonitis, pleuritis, pneumonia, nephritis, bronchitis, iritis, trigeminal neuralgia, sciatica, venous thrombosis, epistaxis and mastitis.

Recurrence and Relapse.—One attack of rheumatism is extremely liable to be followed by later ones, especially in childhood. There is rather the reverse of a protective influence. The severity of the recurrences is in no way dependent upon that of the primary attack.

The tendency for the disease to relapse is very great. These relapses may take place in spite of the greatest care and entirely without discoverable cause, or they may be brought on by slight exposure, such as leaving the bed, a week or so after convalescence seems established. The disease may appear in the joints originally affected or in others. Repeated relapses may occur one after the other, the severity being independent of that of the first attack.

Prognosis.—The prognosis in children is better than in adults so far as recovery from the individual attack is concerned. The course of arthritis is shorter and the symptoms milder. Endocarditis, if slight, is frequently recovered from; if more severe, valvular insufficiency remains but compensation is much more easily acquired in children than in later life. (See Endocarditis, Vol. II, p. 41.) The prognosis of rheumatism on the whole, however, is more serious in early life, on account of the greater tendency to recurrence of arthritis and chorea, and the greater probability that endocarditis will develop in later attacks, if not in the primary one; or that if already present, it will become worse on each reappearance of rheumatic disease. This great disposition to the development of cardiac involvement in early life makes the prognosis of rheumatism in children always doubtful. Rheumatic pericarditis in children is exceedingly liable to lead to loss of cardiac compensation. The studies of Poynton, Agassiz and Taylor¹ upon 350 fatal cases of rheumatism indicated that the disease was at its worst from the 6th to the 12th year, and the large majority of deaths occurred before the age of 20 years. The cause of death in early life was nearly always cardiac involvement.

The prognosis of chronic articular rheumatism is very grave. Though the course of the disease is always slow, it is more rapid than in adults. Some of the milder cases may recover, but this is exceptional. Death finally occurs from exhaustion or some intercurrent disease, especially tuberculosis.

Diagnosis.—Diagnosis in early life is not always easy on account of the different guises under which the disease may appear. The discovery of a slight valvular lesion of the heart should always awaken the suspicion of rheumatism, and an investigation should be made into the previous existence of slight pain in the joints, growing pains, torticollis, repeated sore throat, chorea, and other rheumatic manifestations. So, also, the development of chorea should lead to a careful study of the personal and the family history with regard to the previous occurrence of rheumatism, and to a search for the presence of endocarditis or other rheumatic symptoms.

An attack of acute articular rheumatism is generally easily recognized,

¹ *Loc. cit.*

its characteristic symptoms being sudden development of some degree of pain, swelling, tenderness, and redness in one or, generally, more joints. A number of other conditions may, however, be confounded with it and must be eliminated. *Osteomyelitis* is among these. It is less often multiple in its localization, exhibits more severe constitutional symptoms, and involves the shafts and epiphyses rather than the joint itself. *Secondary arthritis* after acute infectious diseases, such as scarlatina, is usually monarticular, sometimes polyarticular, and is recognized by the previous history of the case. *Gonorrheal arthritis*, although usually monarticular, is more often multiple in early life than later. It is, however, nearly always combined with vulvovaginitis or ophthalmia. The *syphilitic arthritis* sometimes occurring as a later manifestation of syphilis is localized in both knee-joints, runs a very chronic course, and is generally associated with keratitis and other late syphilitic symptoms. *Septic arthritis* is polyarticular, purulent in character, and is always accompanied by other symptoms of sepsis. In infancy *scorbutus* and *syphilitic epiphysitis* are sometimes wrongly called rheumatism; but rheumatism is so rare at this period that it is proper to assume its absence unless its symptoms are unquestionable. Nearly all the cases of pain in the legs in infants, with disability and without fever, are due to scurvy. (See Scurvy, p. 602.) *Multiple neuritis* and *sciatica* may at first suggest rheumatism but exhibit no articular involvement; and the fever and pain on being moved often seen in *poliomyelitis* may cause confusion, but is distinguished by the absence of articular effusion. *Tuberculous coxitis* may also at times occasion difficulty. Careful examination will soon reveal the true condition. *Retropharyngeal abscess* may in some instances simulate torticollis. The course of the case soon renders the diagnosis clear.

Treatment. Prophylaxis.—This is by all odds the most important, since little can be done to influence a cardiac rheumatism when present, and it is the cardiac disease upon which the gravity of the prognosis depends in most cases. Children with a decided family history of rheumatism or who have suffered from previous attacks should be scrupulously guarded against exposure to cold and damp, including wetting of the feet; dressed warmly in woolen underclothing, yet not so warmly that free perspiration is produced; enjoy an abundance of fresh air on suitable days, and live in dry, healthful dwellings. At the same time care must be taken against making the child susceptible by too great precautions. The effort should be made to improve the general tone and to increase the resisting power to influences which might prove harmful. A system of hardening should be cautiously instituted, through cool, morning sponging followed by vigorous rubbing, systematic gymnastic exercises and massage, the fullest use of these measures being approached by degrees. In specially susceptible subjects change of residence in winter to a warm, dry climate is most advisable.

Whether diet has any influence in preventing the disease is disputed. The giving of nitrogenous food and the restriction of carbohydrates has been recommended, but the actual value of these is questionable.

For the prevention of cardiac involvement the cases of even slight articular rheumatism should be kept quiet in a warm room, and, if there is the slightest fever, in bed. This is all that can be done.

Treatment of the Attack.—In cases of acute articular rheumatism the diet during the acute febrile stage should consist of milk or broths. Later a simple, digestible regimen may be prescribed. The patient should be at rest in bed in an equably heated but well-ventilated room, and

the slightest chilling should be avoided since a relapse of the disease may readily follow. The clothing should consequently be preferably of flannel, made easily removable to facilitate changing when wet by the sweating. For local treatment the affected joints may be wrapped in cotton or with warm, moist, applications for the relief of pain; all motion avoided; and the bed-clothes prevented from pressure on tender regions. The treatment of developing endocarditis will be considered in the section upon Tendocarditis, Vol. II, p. 141. For the direct medicinal treatment of the attack salicylic acid has been considered a specific. Whether or not it may be justly deemed so is a matter of much dispute. There is no doubt at least that the salicylates control both pain and fever. To be of value, sufficiently large doses should be given, 5 to 8 grains (0.32 to 0.52) of the salicylate of soda being administered every 3 hours to a child 8 to 10 years of age. Very much larger initial doses are recommended by some clinicians. The dose mentioned can be rapidly increased in size if necessary, and if the stomach tolerates it, until moderate tinnitus and deafness are produced. The addition of an aromatic, such as ginger, often aids in the tolerance of the drug. Salicin, oil of wintergreen, aspirin, salol, and other salicylic acid derivatives, may be given in place of the salicylate; or, if the digestion will not bear any internal administration, the oil of wintergreen (salicylate of methyl) may be used freely by inunction in the armpits and similar suitable regions. The constitutional effect may be produced in this way. When the pain and fever are decidedly lessened the dosage may be reduced, but the treatment should not be abandoned entirely for some time after convalescence seems established. Where the salicylates fail to relieve pain, antipyrine or phenacetin may be employed, alone or in combination with these. For the pain in severe cases opiates may be necessary. I have never witnessed any of the depressing effects from salicylates which have sometimes been reported. The alkaline treatment, as with bicarbonate of soda or citrate of potash, has been much esteemed. It may be combined with the salicylates and should be given in sufficient dose to render the urine alkaline.

Experiments with serum and vaccine treatment have been made, but the results, which have been reported as encouraging, demand further corroborative experience.

During convalescence from acute articular rheumatism tonic remedies may be required, such as arsenic, iron, or cod-liver oil. The treatment of acute manifestations of other forms of rheumatism will be considered in the different chapters discussing these subjects.

In the treatment of subacute and chronic rheumatism the effort must be made to discover the cause. In this direction the condition of the tonsils and mouth should be carefully examined and appropriate measures taken if needed. Autogenous vaccines have been tried with some reputed success. Apart from these methods the persistent administration of iodide of potassium has much in its favor. In other cases arsenic or cod-liver oil is of benefit. Thyroid extract has occasionally given good results. Always the effort to improve the general health must be made and to procure the most favorable hygienic conditions. Massage and passive movement of the joints are very important, except during exacerbations when rest is required. Local applications of ichthyol ointment or preparations of iodine or mercury may be employed. Benefit has followed the production of passive congestion by the Bier method, as well as by baking in a suitable apparatus. Treatment at some one of the thermal springs is of decided help and should always be employed when possible.

CHAPTER VI

THE DIATHESES

The views of many years ago concerning the existence of various diatheses passed to a large extent into the class of forgotten things, under the increasing trend of the attributing of nearly every disordered state to the influence of some infection. In recent years it has become evident that neither infection nor any other active cause is sufficient by itself to account for the development of certain disorders in certain persons; and that diatheses do, in fact, exist. By this term is meant a constitutional tendency to the development of certain sorts of diseases varying with the individual; a tendency which makes the same acting cause vary in the character of the symptoms produced. It is to be noted, however, that in the strict sense a diathesis is not a disease, but a constitutional peculiarity which acts as a predisposition.

Various diatheses have been described, the boundary-lines between them being not sharply marked, and the list of symptoms attributed to their influence varying with different writers. Among them may be mentioned the spasmophilic, lymphatic, neuropathic and exudative. It is further to be observed that there is frequently a combination of two diatheses, the evidences of both appearing in the same individual. The symptoms of the lymphatic diathesis are to a large extent by many attributed to the exudative diathesis, and it is certain that there exists in any event a close relationship between them. Whether they should, in fact, be classed as one is not yet possible of determination. Spasmophilia is by many considered as one form of the neuropathic diathesis, and its occurrence in combination with the exudative diathesis is very common; while in other cases lymphatic disturbances occurring in nervous subjects give rise to the title "neurolymphatic diathesis." All this renders the subject confusing and the divisions arbitrary and conflicting.

The spasmophilic and neuropathic diatheses are described elsewhere (see Vol. II, pp. 249 and 269) in connection with Diseases of the Nervous System. Here will be considered only the exudative and the lymphatic diatheses.

EXUDATIVE DIATHESIS

Etiology.—This title was applied by Czerny¹ to subjects in which, with other symptoms, there is a tendency to *exudation*, or inflammation, of the skin and mucous membrane. It covers many of the symptoms formerly described as "scrofulous," but is in no way connected etiologically with tuberculosis. It occurs chiefly in the 1st year of life, but to a lesser extent after this period. It is to a considerable extent hereditary and familial, several children of the family exhibiting the same symptoms, and the parents perhaps having suffered from eczema, gout, asthma, or some nervous disorder. Apart from the constitutional tendency, the symptoms are brought on or increased by an improper diet, such as one containing an excess of food of any sort, especially one too largely of milk, and, most of all, of fat.

Symptoms.—The disease manifests itself in infancy by an unusual disposition to the development of seborrhea of the scalp and face, and later of eczema; the latter often extending over much of the body. Catarrhal inflammation of the nose and pharynx, as well as bronchitis,

¹ Jahrb. f. Kinderh., 1905, LXI, 199.

is common, and otitis a frequent consequence. Some enlargement of the neighboring lymphatic glands may be found, but decided and extensive hypertrophy is not a characteristic of this disease. The infants are usually plump, but flabby and anemic. Elevation of temperature occurs readily. In other cases they are thin even before symptoms appear, or become debilitated and lose flesh through the constant irritation and loss of sleep brought about by the itching of an eczema. The blood shows an increase of the eosinophilic cells, perhaps up to 10 or 20 per cent. Whether this depends upon eczema, of this is present, or occurs coincidentally and from the same cause, is uncertain. The fact that it is seen in asthma also is suggestive of the latter relationship. It has been claimed that the sugar-content of the blood and the retention of chlorides is increased, but this demands further proof.

In older children, after the period of infancy, eczema may be replaced by asthma, obstinate cough, pruritis, lichen, and urticaria. The geographical tongue is a common symptom. Vasomotor disturbances are frequent, such as palpitation, rises of temperature, and the like. It is uncertain whether the phlyctenules which appear on the conjunctiva in children are symptoms of the exudative diathesis, or are actual tuberculous lesions.

In the category of the exudative diathesis perhaps belong, too, many of the cases of *arthritis* in children, described by Comby¹ and others. The symptoms are variable and multiform. Prominent among them are those just detailed, in addition to attacks of vomiting (recurrent vomiting) and various other nervous and vasomotor disturbances.

Course and Prognosis.—The tendency to eczema diminishes greatly after the 1st year, and in general the exudative symptoms are prone to ameliorate and soon to cease. Sometimes, however, the symptoms characteristic of the condition after this period are very slow in disappearing. Generally they are gone by the time puberty is reached. They do not often in themselves constitute an element of danger. Fatal results generally depend upon an attendant spasmophilic or lymphatic diathesis.

Diagnosis.—This rests upon the symptoms as already outlined. Scrofulo-tuberculosis may, it is true, develop in children with the exudative diathesis, but the eczema and catarrhal condition of the latter has nothing in common with the glandular inflammation, chronic conjunctivitis and keratitis, and severe chronic catarrhal processes of the former; although it is possible that it is the existence of the exudative condition which predisposes to a tuberculous infection producing the symptoms often called scrofula. Similarly the status lymphaticus is characterized by decided hypertrophy of the lymphatic tissues throughout the body, including the thymus gland, and by a tendency to sudden death; but it is not a producer of eczema or catarrhal processes. It is, however, frequently combined with the exudative diathesis.

Treatment.—Only the passing of time will cure the constitutional predisposition, and treatment must be directed against the immediate exciting cause of the symptoms. This is chiefly dietetic. The fat in the food must be reduced, and indeed the total amount of food given usually diminished as well, and care taken to prevent the children from gaining weight rapidly. Starchy addition to the diet should be commenced at once on the appearance of symptoms, and this made to replace milk to a considerable extent.

¹ Arch. de. méd. des enf., 1902, V, 1; 65.

LYMPHATIC DIATHESIS

(Lymphatism. Status Lymphaticus)

This is a disorder much discussed and not yet entirely understood. It would appear to be a constitutional condition in which there is present a diminished resistance of the entire organism to morbid influences, and a certain hypersensitiveness of the nervous system producing a predisposition to sudden death from cardiac failure brought on by slight and in themselves insufficient causes, or occurring entirely unexpectedly and apparently without reason. Anatomically there is a tendency to general hyperplasia of the lymphoid tissues throughout the body, including the thymus gland. The close association of lymphatism with sudden death and thymic enlargement was emphasized by Paltauf;¹ while Escherich² maintained that the condition was a toxemia, the origin of which was the hypertrophied thymus gland. The relative relationship of thymic hypertrophy and of general lymphatic enlargement is not yet clear. It is possible that lymphatic overgrowth is the direct cause of the symptoms, and that the enlargement of the thymus gland is an accidental or a compensatory process. Whether, however, a lymphoid hyperplasia is a necessary condition, or even one always present has been disputed; and the theory has been advanced by Heubner³ and others, and has much in its favor, that the disorder is a constitutional anomaly consequent upon some chemical alteration of the tissues, independent of lymphatic or thymic enlargement, although these may appear among the later symptoms. Personal experience has led me to the belief that the enlargement of the thymus, and perhaps too of the lymphatic glands, is a secondary matter, and can be entirely absent in cases of sudden death, and present in those dying of other causes. (See also Sudden Death, p. 216, and Enlargement of the Thymus, Vol. II, p. 518.)

There are cases with glandular enlargement and symptoms of other sorts to which the title "scrofulous" was formerly, and is still often, applied, the lesions being in reality tuberculous manifestations, although the constitutional condition would place the child in the class of those suffering from the lymphatic or oftener from the exudative diathesis (p. 630. See also Tuberculosis, p. 559, and Adenitis, Vol. II, p. 249.) There is an undoubted close relation between lymphatism and spasmophilia (see Vol. II, p. 249) on the one hand, and the exudative diathesis on the other.

Etiology.—A distinct family disposition is seen in many instances. A tendency to glandular swelling may have existed in the parents as well as the children; and as regards the most severe form, I have previously reported⁴ the occurrence of 9 sudden deaths from lymphatism in one family of children. The disease is observed especially in infancy and early childhood, although in the disposition to sudden death from slight causes it has not infrequently been witnessed in adult life. Even the new born and very young infants may exhibit it; and many instances have been wrongly attributed to death from suffocation through overlying or brought about in other ways. Sex, race and season exert no influence. Rickets is often associated with the lymphatic diathesis, but has no etiological connection, other than that both may possibly be produced by allied causes acting simultaneously and consequently combined; and

¹ Wien. klin. Wochenschr., 1889, No. 46; 1890, No. 9.

² Berliner klin. Wochenschr., 1896, XXXIII, 645.

³ Kinderheilkunde, 1911, 33.

⁴ New York Med. Journ., 1909, Sept. 4.

the same is true of spasmophilia and of the exudative diathesis. The constitutional tendency may be present at birth, or may be acquired especially through errors in hygiene and diet; or may disappear under regulation of these or perhaps as a result of increasing age.

Pathological Anatomy.—The noteworthy feature often found at autopsy is the hyperplasia of lymphoid tissue. This is seen especially in the thymus gland, which is frequently abnormally large. In determining the existence of enlargement the great variation in the size of the normal thymus gland in different children is to be taken into account, as well as the age of the patient. (See Physiology, p. 62; Diseases of the Thymus Gland, Vol. II, p. 518.) In addition to this lesion there is commonly more or less hypertrophy of the lymphoid tissue throughout the body, particularly well seen in the glands of the neck and head, and in the tracheo-bronchial and mesenteric glands, as also in the nasopharynx, the posterior pharyngeal wall, the base of the tongue, the tonsils, the entrance to the larynx, Peyer's patches, and the solitary follicles of the intestine. There is usually moderate enlargement of the spleen, with prominence of the Malpighian bodies. The liver is often fatty and the cardiac muscle may show degeneration. In determining the presence of abnormal lymphatic overgrowth, the natural tendency of all normal children to a certain degree of this is to be taken into account.

Symptoms.—These are often vague or even unnoticed; often sufficiently well marked, but varied. The frequent association of the exudative diathesis accounts for the tendency to seborrhea and eczema so often seen. Lymphoid hypertrophy is generally discovered in some part of the body. It is often and sometimes early shown in the development of adenoid growths of the nasopharynx, or increase of lymphoid tissue at the base of the tongue or on the posterior pharyngeal wall. In other cases there may be discovered persistent and sometimes decided enlargement of the lymphatic glands in the region of the neck, occiput, axillæ, or groins. The spleen is often found enlarged on palpation, and it may be possible to detect by percussion or by radiography, or occasionally by palpation, an enlargement of the thymus gland. Generally, however, thymic enlargement cannot be satisfactorily determined during life. (See Diseases of the Thymus Gland, Vol. II, p. 518.) Other cases show a tendency to asthmatic respiration or to attacks of asphyxia dependent sometimes upon pressure of an enlarged thymus gland; much oftener upon the nervous disturbance. Ordinary asthmatic bronchitis is a symptom rather of the exudative diathesis. The combination of the neuro-pathic diathesis or of spasmophilia frequently observed accounts for the occurrence of convulsions, dyspnea, fever from insignificant causes, and laryngospasm; but it is not certain how often this is the case and how many instances, especially of the last mentioned, depend purely upon lymphatism. The children are usually phlegmatic, flabby, fat, inactive, pale, and of little strength. There is a very notably diminished resisting power and increased susceptibility to disease, and infections of any sort may produce an unusually well-marked reaction.

Of all symptoms, however, the most important is the danger of sudden death. This often appears to be respiratory from asphyxia and cyanosis, but probably in most cases is cardiac, as was pointed out by Pott,¹ the accident occurring without discoverable reason; or following very insignificant trauma, such as a hypodermic injection, an exploratory

¹ Jahrb. f. Kinderh., 1892, XXXIV, 118.

puncture, the giving of diphtheria antitoxin, and the like; or being produced by the shock from cold water, the use of a tongue-depressor, the administration of an anesthetic, or the occurrence of some mild acute disease; or often without there having been any previous symptoms whatever. In any event the patient suffers from a sudden, apparently suffocative attack, throws the head back, turns pale, or perhaps bluish, and perhaps dies in an instant; or the child may possibly be found dead in bed without any symptoms having been observed. Sometimes the death is not entirely without warning, and attacks which appear to be laryngospasm, or symptoms suggesting asphyxia from other causes, may be present for several hours. In still other instances there may have been a series of short attacks of the nature described occurring during some weeks or longer, and the patient at last dies in one of these. I have known such short attacks to be of very great frequency. Thus in one case the mother, a foreigner with little command of English, said of her child that it "died every day." Probably the majority of cases of lymphatism of this severest form are not recognized until a sudden fatal attack occurs.

Prognosis.—For the milder cases the prognosis is on the whole good, if dangerous symptoms can be avoided. Under proper management the evidences of the disease may be kept in abeyance, and even the pathological tendency made to disappear as the patient grows older, and particularly as puberty is approached. On the other hand, the prognosis is always doubtful in individual cases, because of the possibility of the occurrence of sudden death.

Diagnosis.—This can be made if, with the symptoms described, the distinct evidences of lymphatic hyperplasia are discoverable. An x-ray examination may show hypertrophy of the thymus gland. Discovery by the same means of enlarged tracheo-bronchial lymph-nodes does not determine whether these are dependent upon tuberculosis or upon the lymphatic diathesis. Similarly the hypertrophy of the tonsillar tissue of the fauces and nasopharynx is not by itself sufficient to warrant a diagnosis of lymphatism, although it is a characteristic of this condition. Often, in fact, lymphatism is entirely unsuspected until sudden death occurs and autopsy shows general hypertrophy of the lymphatic tissue of the body.

Treatment.—The only treatment possible is the careful regulation of the diet and hygiene, inasmuch as it is very probable that these, and particularly the diet, have a decided etiological influence. There is no rule, however, which indicates what the alteration in the diet should be, and possible faults should be sought for in each individual case. Gymnastic exercises and massage may improve the general health. Suitable tonics may be employed to increase the patient's strength and remove anemia. Whenever the lymphatic diathesis is suspected, great care must be taken to avoid the causes which bring about a sudden fatal termination. The use of an anesthetic must be refrained from if it can be avoided, and, indeed, no operation performed which can in any way be omitted. Cool bathing may be dangerous, as may any other sudden physical or mental shock. Even the giving of antitoxin is to be regarded with anxiety, and the objections to it must be carefully weighed before a decision is reached.

CHAPTER VII

ACIDOSIS

By this term is designated the condition in which acid-substances are present in the blood in a quantity relatively so increased, that the normal excess of alkali is much diminished. Experimentally acidosis with its symptoms has been caused in rabbits by the administration of hydrochloric acid in large amounts. In man it depends upon a relative excess of acid produced in the economy. These acids are in some cases represented by the acetone bodies, and consist of β -oxybutyric acid, acetoacetic acid, and acetone. In other instances, as in the acidosis of diarrhea in infancy, it is certain that these bodies play no part whatever, and that there is little or no increase of them in the blood. Acid-substances of other nature are operative here.

The relative excess of acid in the blood may be due either to an overproduction of acid-bodies; to a failure of the lungs and kidneys to excrete them in sufficient amount; or possibly to a loss of bases from the body (Howland and Marriott).¹ Thus the removal of alkali from the system might produce acidosis, as well as the formation of an excess of acids.

The acetone-bodies are normally present in small amount in the blood and urine of healthy children, as shown, among others, by Moore² and by Veeder and Johnson.³ The amount is increased in starvation, acute febrile diseases, severe diarrhea, diabetes, intestinal autointoxication, and recurrent vomiting. The presence of an excess of these in the urine may or may not be accompanied by symptoms of acidosis. The term "acidosis" denotes a decrease in the alkalinity of the blood, as indicated by diminished carbonic dioxide tension of the alveolar air, an increase in the hydrogen-ion concentration of the blood, a great lessening of the alkali reserve of the blood, and an increase of the ammonia of the urine. These constitute a disturbance of the normal balance between the acid and the alkali of the blood.

A sharp distinction must therefore be drawn between acetonuria and acidosis. This is necessary on account of the wide-spread confusion which has arisen, according to which acidosis is supposed to be always indicated by acetonuria. *Acetonuria* consists merely in the presence of acetone-bodies in the urine. It is a matter of excretion, and may or may not be accompanied by symptoms of acidosis. *Acidosis*, on the other hand, as has been stated, is characterized by a relative decrease in the alkalinity of the blood, independent of the amount of the acetone bodies which is being excreted. It may occur without the presence of any of these bodies in the urine, and may depend upon other acid-substances; possibly acid phosphate, lactic acid, or the like. It is only by the presence of characteristic symptoms and by laboratory tests that the diagnosis of acidosis can be made with certainty. Errors in diagnosis are frequent, on account of the misconception of this contrast. Any starving child, as, for instance, one with repeated vomiting and the consequent emptiness of the stomach, exhibits an increased amount of the acetone-bodies in the urine; and this has been wrongly assumed to be the proof of the existence of acidosis. So, too, a large proportion of

¹ Amer. Journ. Dis. Child., 1916, XI, 309.

² Amer. Journ. Dis. Child., 1916, XI, 211.

³ Amer. Journ. Dis. Child., 1916, XI, 291.

cases of acute febrile disorders in early life exhibit a decided acetonuria, but without suffering in any way from acidosis.

On the other hand, it is certainly true that children are especially predisposed to the development of acidosis. This depends either upon the smaller reserve of alkali which they possess, or upon the readiness with which their organism produces the acetone-bodies, without any discoverable explanation.

Acidosis is a common symptom in diabetes at any age. In early life this disease is a less frequent etiological factor merely on account of its less frequent occurrence; since a most dangerous acidosis develops with the greatest suddenness and facility in diabetes in children. A much more common cause is severe diarrhea, especially of the class of "food intoxication," "cholera infantum," "summer diarrhea," and the like, in which rapid and profuse loss of liquid and of the bases of the body takes place from the intestine. This is a form of the disease clearly not dependent upon the production of any excess of the acetone-bodies in the blood. Just what etiological relationship the retention of acid phosphate may possess appears uncertain.

Acetonuria is a frequent symptom, too, in recurrent vomiting, but would appear to be oftener a result of the starvation than an indication that acidosis is the cause of this disease, especially as vomiting is not a characteristic symptom of acidosis. The exact relationship of acidosis to recurrent vomiting does not as yet appear to be fully determined. (See *Recurrent Vomiting*, p. 701.) The disease is apparently due to some intoxication, and this may be an acidosis, but the presence of acetonuria is no proof of it.

Pneumonia and nephritis may be productive of acidosis, and it is very probable that many of the symptoms denominated uremic may be, in reality, dependent upon an acid intoxication. Finally severe acidosis may occasionally develop without any discoverable reason whatever.

There are certain **symptoms** which are suggestive of the disorder. Among the earliest of these may be mentioned restlessness, sleeplessness, excitement; and later a tendency to prostration, somnolence, and coma. When these appear in a case of diabetes, or in an infant with severe summer diarrhea, and decided oliguria, acidosis can with reason be suspected. The only positive symptom, however, apart from laboratory tests, is hyperpnea. This consists in a remarkable alteration of respiration, with deep and exaggerated inspiration and expiration, usually not increased in rapidity, and constantly present; yet without any functional or organic disorder of the lungs or heart to account for this, and without cyanosis. The degree of hyperpnea is directly proportional to the reduction of the alkali reserve of the blood, as shown by laboratory tests.

The **prognosis** is in most cases unfavorable. If once the symptoms are well developed, death is liable to occur. The relief of coma and of hyperpnea may be effected at least temporarily, but with only a deferring of the fatal issue. Coma may return and death result without the redevelopment of any urinary symptoms to account for it.

The **treatment** should be *preventive*. If any diseased condition exists in which it is known that there is danger of acidosis developing, the occurrence of the symptoms must be carefully guarded against. In diabetes the greatest caution must be observed in the removal of the carbohydrates from the diet, without a simultaneous cutting off of the protein, and especially of the fat. Starvation is the procedure indicated for the beginning of treatment. In the case of severe, profuse, diarrheal discharges in infancy,

prompt means must be taken to check the loss of liquid from the system. Initial purgation in such cases is dangerous, and astringents and opium should be used at once, unless the presence of fever, tympanites, and the character of the stools indicate that irritating substances are still present in the intestinal canal. In all cases where acidosis is feared there should be a prompt administration of alkali, especially bicarbonate of soda, in sufficient amount to render the urine alkaline. It may be given by the mouth or by the bowel. Even in cases where well-marked symptoms of acidosis are already present, efforts should be made to restore the alkali reserve of the blood, the production of alkalinity of the urine being the indication of the success attained. In normal infants 2 to 3 grams (0.07 to 0.11 oz.) of bicarbonate of soda is sufficient to render the urine alkaline; but when acidosis is present from 4 to 10 times this amount may be required (Howland and Marriott).¹ In urgent cases it is best to administer the soda intravenously, using a 4 per cent. solution, and giving at one time 75 c.c. (2.5 fl. oz.), or more in infancy; the determination of the amount and of the number of injections being decided by the production of urinary alkalinity. The solution for this purpose should be prepared by a special method, since the process of sterilization decomposes the sodium bicarbonate and renders the solution very irritating. (See p. 232.) In spite of care, however, sloughing of the tissues may occur, if any of the fluid escapes into them. The subcutaneous injection of the fluid is to be deprecated, as damage to the tissues is extremely likely to follow, even with the specially prepared solution. It is true that there is danger of the administration of alkali in excess, even when checked by repeated examinations of the reaction of the urine; but the danger appears to be decidedly less than that caused by the persistence of the acidosis.

In addition to the treatment by alkalis the administration of water in large amount is important in all cases, and especially in those in which there has been a large loss of liquid from the system through the presence of diarrhea. If this cannot be given by the mouth, or if not retained by the rectum, it may be administered in the form of normal saline solution, either by hypodermoclysis or, better, by intraperitoneal injection.

CHAPTER VIII

DIABETES MELLITUS

Only the salient features, especially as applied to children, can be discussed in this connection. Although known to the ancients it was first clearly distinguished from diabetes insipidus by Willis.² Although much less frequent in early life than later, my own experience accords with that of those who find it occurring at this time much oftener than formerly supposed, or than many statistics would indicate. It is to be distinguished from alimentary glycosuria and transitory glycosuria from other causes. (See Vol. II, p. 168.) Very probably the statistics of diabetes in children include many instances which belong to this class of cases. A characteristic distinction is that pointed out by Allen,³ that whereas in non-diabetics the ingestion of carbohydrates in excess increases the amount which can be

¹ *Loc. cit.*

² *Pharmaceut. ration.*, 1674, Sect. IV, Cap. 111. Ref., Senator in Ziemssen's *Handb. d. spec. Path. u. Therap.*, 1867, XIII, 2, 117.

³ *Glycosuria and Diabetes*, 1913, 1050.

utilized by the subject, in diabetics the tolerance diminishes in proportion as the amount of carbohydrate is increased.

Etiology.—*Age* is an important factor. von Noorden,¹ found in 3000 cases of diabetes 2.8 per cent. occurring in the first 10 years of life. The report of the Registrar General for England and Wales from 1861 to 1870 inclusive gave a very similar percentage (Dickinson).² In 6494 deaths from diabetes there were:

TABLE 79.—MORTALITY FROM DIABETES IN EARLY LIFE,
ENGLAND AND WALES

Under 1 year.....	8
1 year old.....	19
2 years old.....	16
3 years old.....	15
4 years old.....	16
Total under 5 years.....	74 (1.1%)
5 to 10 years.....	114
Total under 10 years.....	188 (2.9%)
10 to 15 years.....	200 (3.0%)

Joslin's³ statistics show a larger number; 4.7 per cent. of his 1156 cases having commenced in the first 10 years of life. The majority of deaths in early life occur after the age of 5 years, and especially from 10 to 14 years of age. The statistics given show, however, that infants even in the 1st year are attacked. The youngest cases under my care were in girls of 15 and 16½ months respectively. Knox⁴ collected 16 cases in the 1st year of life. In Stern's statistics⁵ of 117 collected cases in children, 6 were under 1 year of age, and in 1 of these the disease appeared to be congenital. Such a case appears to be that of Cuno⁶ of pancreatic diabetes in a child of 3 weeks. In Wegeli's⁷ 108 collected cases in children, not included in Stern's series, there were:

TABLE 80.—INCIDENCE OF DIABETES IN EARLY LIFE

Under 1 year.....	3 cases
1 to 5 years.....	26 cases
5 to 10 years.....	31 cases
10 to 16 years.....	42 cases

In adult life diabetes is distinctly more common in males than in females, but in children sex appears to have but little influence. *Heredity* plays a very important part, the disease having shown itself in the parents or grandparents, or more than one of the children being attacked. This is true in from 10 to 25 per cent. of the published statistics. Consanguinity, gout, nervous disorders, tuberculosis and syphilis of the parents have also been considered causes. Trauma of the head and intracranial tumors, especially of the medulla, have been followed by the disease. An excessive consumption of sugar sometimes results in a permanent glycosuria. Diabetes occasionally follows acute diseases or nervous shock. Probably, however, the majority of cases are associated with lesions or disturbance of function of the pancreas.

¹ Pfaundler u Schlossmann, Handb. d. Kinderheilk., 1910, II, 117.

² Diabetes, 1875, 66.

³ Treatment of Diabetes Mellitus, 1917, 28.

⁴ Johns Hopkins Hosp. Bull., 1913, XXIV, 274.

⁵ Arch. f. Kinderh., 1889-90, XI, 82.

⁶ Jahrb. f. Kinderh., 1910, LXXI, 623.

⁷ Archiv f. Kinderh., 1895, XIX, 1.

Symptoms.—These do not differ materially from those in adult life, except that the onset is frequently more rapid and the course shorter. The chief symptoms consist in wasting, thirst, great appetite, and excessive secretion of urine containing glucose. In the beginning, and perhaps during several months, the symptoms are but little marked, and only unusual thirst or abundant secretion of urine, or moderate loss of weight and strength are noticed. The child does not seem ill in other respects and is decidedly improved by dietetic treatment. After a variable time the symptoms increase in severity. The thirst is very great and a surprising amount of water is drunk; the appetite is sometimes voracious, sometimes diminished; the tongue is red and dry; the teeth decay readily; the skin is dry and harsh and usually somewhat pale, dependent upon the anemia which is generally present. Perspiration is diminished or absent. Constipation is frequent. Nocturnal incontinence of urine is a common and often an early symptom. There is decided debility and the patient becomes very easily exhausted and suffers from loss of spirits, irritability, headache and diffuse pains. Wasting is progressive and rapid and finally becomes extreme.

As regards the character of the urine, the total secretion often reaches 3 or 4 times the normal amount, exceptionally even equalling 6 or 8 quarts (5672 or 7563) in 24 hours. It is pale in color, acid, and with the specific gravity ranging from 1025 to even 1050, although occasionally it may not be over 1015. The percentage of sugar varies from a small amount up to 5 per cent. or more, and even as much as 10 or 12 per cent. has been reported. The total quantity of sugar excreted in 24 hours may reach 6 ounces (170) or over. A small amount of albumin is frequent with a few casts. Acidosis is a characteristic of the disease, seen in all advanced cases. As it develops, acetone, diacetic acid and β -oxybutyric acid appear in the urine.

Complications.—These are much less frequent than in adult life. Epistaxis, furunculosis, urticaria, erythema, cutaneous abscesses, and eczema are not uncommon. Gangrene is rarely seen in early life. Cataract has been reported, but is not common at this age. This is true also of optic neuritis and retinitis. Nephritis of moderate degree is not infrequent. Peripheral neuritis and otitis are sometimes seen. Tuberculosis, or, oftener, bronchopneumonia, is a not unusual complication.

Course and Prognosis.—The course of diabetes in children is usually more rapid than in adults. It is rare that the attack lasts much over a year from the time symptoms are discovered, and usually a shorter time. As the earlier course of the disease is seldom recognized, the total duration of the affection is probably considerably longer than supposed. Some of the acute cases have apparently lasted only a few weeks. I have seen one instance in a girl of 4 years in which the interval from apparently perfect health without symptoms of any sort to death in coma equalled only 11 days. Under ordinary circumstances the course is progressive if no intercurrent disease occurs to terminate life. Weakness and emaciation increase and finally coma develops. Death in coma is especially frequent in children; that from complications much less often seen than in adults. Generally the approach of coma is indicated by loss of appetite, nausea, vomiting, epigastric pain, the presence of acetone in the urine and its odor on the breath, great weakness and nervous irritability with jactitation, or somnolence. Unconsciousness then comes on with its characteristic deep respiration. Under treatment the fatal issue may be delayed, but if coma has developed death

is almost certain within a few days. The final prognosis in children is very unfavorable, if care be taken to exclude from the list cases not certainly diabetes, such as instances of transitory or alimentary glycosuria. Yet under newer methods of treatment the prognosis does not appear to be so uniformly bad as was formerly considered to be the case, and it would appear, as pointed out by Riesman¹ and others, that the disease at this time of life may sometimes run a mild course and terminate in recovery.

Diagnosis.—The disease would doubtless be oftener recognized were the urine of infants and children examined more systematically. The diagnosis rests principally upon the dryness of the skin, thirst, normal or increased appetite, loss of weight and strength, enuresis, increased secretion of urine and the recognition of grape sugar in it.

Diabetes is especially to be distinguished from other conditions in which some form of sugar is present in the urine. *Transitory glycosuria* following gastro-intestinal disturbance or attacks of infectious diseases may resemble it closely. It lasts, however, but a few weeks at most and is not attended by the characteristic symptoms of diabetes. *Alimentary glycosuria* also may occur in certain individuals when an unusual amount of sugar is contained in the food. In the case of infants lactose may sometimes appear in the urine under similar conditions. The reduction of the amount of sugar given is followed immediately by disappearance of the symptom. It must be remembered, too, that there are other substances besides sugar which will reduce the copper of Fehling's solution. Other tests, such as fermentation, phenylhydrazin, or the use of the polariscope, must be employed before a positive diagnosis can be given in some cases. I have seen much unnecessary alarm caused by a too hasty diagnosis.

Treatment.—This is much the same as in adult life and need not be entered upon in full detail. It is more unsatisfactory in children, partly on account of the greater severity of the disease as usually seen at this time, partly owing to the difficulty in obtaining the coöperation of the child in following a strict diabetic diet. The purpose of treatment is to remove the strain imposed by the secretion of sugar, and thus reëstablish the lost tolerance for carbohydrates; the patient whose urine has been made sugar-free having the carbohydrates then gradually and cautiously increased, according to the degree of tolerance obtained. A difficulty, especially in children, in the total withdrawal of carbohydrates while protein and fat are continued, is that a condition of dangerous acidosis, even with the development of coma, may be promptly produced in those who had not seemed in danger of this condition. The plan of treatment proposed by Allen² has been remarkably successful in the experience of various clinicians. It consists in beginning with an almost complete starvation, giving only water, clear broth, and, if necessary, a small amount of whiskey; this last especially if acidosis is present. This may be continued for not more than 3 days, by which time, in favorable cases, the urine has become sugar-free. This fasting, which would produce acetoneuria in normal individuals, relieves it in diabetes. Now small amounts of food are cautiously given, preferably green vegetables, such as spinach, asparagus, lettuce, celery, and the like, containing from 5 to 6 per cent. of carbohydrate (see Table by Joslin),³ and after this protein,

¹ Amer. Journ. Med. Sci., 1916, CLI, 40.

² Bost. Med. and Surg. Journ., 1915, CLXXII, 241.

³ Amer. Journ. Med. Sci., 1915, CL, 493.

at first in small amounts, and then fat. Not more than from 5 to 10 grams (0.18 to 0.35 oz.) of carbohydrate daily should be allowed at first, and every increase be made cautiously. The protein, too, should be at first in an amount of not over 20 grams (0.71 oz.) per day. Frequent examinations of the urine for sugar and for the acetone bodies are required, to determine the tolerance for carbohydrates. Once a week a day of complete or partial fasting should be ordered, even if the urine is sugar-free. There will be at first a loss of weight, and later but little gain may take place; but this is a matter of entirely secondary consideration. The maintaining of a complete tolerance for carbohydrates is the thing desired. Should sugar appear in the urine the starvation must again be instituted; but this need not continue longer than 24 hours, after which a cautious return to food should be made, as at the beginning. Oatmeal has been recommended by Noorden¹ as one of the best of the cereal foods.

In the case of infants increased difficulty arises owing to the need of milk in the diet. Sour milk may be employed on account of the diminished sugar-content; or buttermilk for the same reason, and because the diminished amount of fat aids in preventing the development of acidosis. Albumen water may form part of the diet.

With regard to the use of drugs, those most in vogue are opium, arsenic, antipyrine, salicylic acid, and Carlsbad water. Amelioration appears to follow in some instances, but the treatment of this disease is not by medicaments. For the prevention or treatment of diabetic coma, the administration of large doses of bicarbonate of soda is advised, 3 to 4 ounces (93 to 124) or more being given daily. This will sometimes overcome the coma for a time, if not severe. Hypodermoclysis, or intravenous injection with 3 per cent. bicarbonate of soda solution is also recommended for the same purpose. The latter must be specially prepared, as it cannot be sterilized without undergoing chemical changes. (See p. 232.)

There is nothing gained by keeping the children in bed, Joslin's² experience in 59 cases of the disease in early life was distinctly in favor of allowing them to be about and to exercise freely. If lack of strength renders rest in bed necessary massage should be employed.

CHAPTER IX

DIABETES INSIPIDUS

Increased secretion of urine, temporary or of a more chronic nature, is of common occurrence, and is due to various causes. This *polyuria* is referred to briefly later. (See Vol. II, p. 164.) Such cases are to be distinguished from diabetes insipidus, in which there is a lasting excessive secretion of urine with great thirst. This condition is not common at any time of life. It was first clearly distinguished from diabetes mellitus by Willis.³

Etiology.—Age exerts no noteworthy predisposing influence. Of 77 cases of all ages collected by Roberts,⁴ 22 (31.4 per cent.) were under

¹ Berl. klin. Wochenschr., 1903, XL, 817.

² *Loc. cit.*, 462.

³ Pharmaceut. ration., 1674, Sec. IV, Cap. III. Ref. Senator, in Ziemssen's Hand. d. spec. Path. u. Therap., 1876, XIII, 2, 254.

⁴ Urinary and Renal Diseases, 3d Amer. Ed., 1879, 198.

10 years of age, and 7 of these under 5 years. In Strauss's¹ series of 85 cases, 21 (24.7 per cent.) were less than 10 years of age, and 9 less than 5 years, and in 124 cases analyzed by Stoermer,² 19 (15.3 per cent.) were under 10 years of age and 12 of these less than 5 years. The disease may appear occasionally to be congenital. In children the influence of sex is inconsiderable, whereas in adults many more males are affected. Inheritance is probably the most powerful predisposing factor, several members of a family perhaps being affected. Various nervous conditions in the parents seem to predispose to the disease in the offspring. Trauma especially of the back of the head or neck has been followed by diabetes insipidus, and this is true also of morbid growths and other diseases of the brain, syphilis, and acute infectious disorders. There has been observed, too, an undoubted connection between diabetes insipidus and the pituitary body, and tumors or diseases of this gland may be attended by polyuria. In the majority of instances, however, no cause can be discovered, although the influence of the pituitary body may still be active although unproven. There are no post-mortem lesions except those productive of the disease in secondary cases.

Symptoms.—The most marked early symptoms are great thirst and excessive secretion of urine. The amount of fluid imbibed is sometimes remarkable and the daily secretion of urine may much exceed that of diabetes mellitus, reaching from 5 to 15 quarts (4732 to 14195) or occasionally more. It is pale, with a specific gravity of from about 1001 to 1005. There is no sugar or albumin present, but in some cases inosite has been found. The total solids excreted are usually normal, sometimes increased. Most patients pass more urine during the night than during the day, and nocturnal enuresis is common. The skin is pale and dry with little or no perspiration; the temperature is sometimes subnormal; the blood exhibits nothing characteristic; the appetite is variable. The general health is often but little affected. In many cases, however, there is more or less emaciation and malnutrition, and bodily and, less often, mental development may be retarded and a degree of infantilism result. Nervous symptoms may be present, among them neuralgias of various kinds, restlessness, vasomotor disorders, and anorexia or excessive appetite.

Course and Prognosis.—The course of the disease is of an essentially chronic nature. Unless terminated by some intercurrent disorder it may last for years;—sometimes from infancy to adult life. In other cases emaciation and debility progress and death ensues from this cause. Recovery, or, in other instances, tolerance may be finally established, but absolute recovery in well-established cases is rare.

Diagnosis.—This rests upon the persistent polyuria and thirst. After infectious fevers there is often a brief transitory polyuria, which lasts a few days or even weeks, and a similar condition may be associated with hysterical or other nervous symptoms. Chronic interstitial nephritis produces polyuria but not to so great a degree. It exhibits albuminuria and casts, with diminution in the excretion of solids, and is attended by other characteristic symptoms.

Treatment.—The principal treatment consists in a careful and gradual restriction of the amount of fluid allowed, not, however, to the extent of producing decided discomfort. The clothing should be warm,

¹ Dissert. Tübingen, 1870. Ref. D. Gerhardt, Nothnagel Spec. Path. u. Therap., VII, 1.

² Dissert. Kiel, 1892. Ref. D. Gerhardt, *loc. cit.*

abundance of fresh air given, and the diet of the most sustaining nature. Very many drugs have been tried, none of them proving satisfactory, among those most in vogue being ergot, antipyrine, arsenic, belladonna and opium. It is doubtful whether the meager hope of benefit from any of these counterbalances the harmful secondary effects which they may produce. That the disease may sometimes be associated with syphilis renders antisypilitic treatment advisable in cases giving a positive Wassermann reaction. More recently pituitrin has been regarded as a specific for the affection. Galvanization of the spinal cord is claimed to have given good results.

CHAPTER X

PELLAGRA

The nature of pellagra (*pelle—agra*, rough skin) is still uncertain. Long attributed almost universally to a food-intoxication, the opinion gradually spread that it was an infectious disease, although satisfactory proof of this had not been supplied. More recently the view has developed that it is a "deficiency" disorder due to an absence of certain necessary substances from the diet. It was first recognized as a morbid entity by Gasper Casal in Spain¹ in 1735, although it had previously been known under various popular names; among them that of "Mal de la Rosa." For many years it has been of frequent occurrence in Italy, Roumania, Austria and some other southern countries of Europe, as well as in Egypt and India. Some cases have been reported in England and Scotland. It had been observed from time to time in the United States for a good many years, a case having been reported by Babcock² occurring as long ago as 1834, and one by Gray³ in 1864. Since 1906 it has been recognized with alarmingly increasing frequency. It was estimated by Lavinder⁴ that over 30,000 cases had occurred in this country, chiefly in the Southern States; yet nearly every State of the Union has suffered from it. Several cases in Philadelphia, have come under my observation 2 of them in children who had never been beyond the borders of Pennsylvania. The disease, or at any rate the recognition of it, is certainly extending with great rapidity.

Etiology.—Pellagra attacks all ages, although it seems most common in young adults. Studies of the malady as it is found in children in the United States have been made by Snyder,⁵ Weston⁶ and Rice.⁷ These show that it may occur even as early as 2 months of life, and that it is rather frequent between the ages of 6 and 18 months. In 323 cases studied personally by Grimm,⁸ 15 per cent. were in subjects in the first 15 years of life. It is very probable that the incidence in childhood is much greater than this. Females are oftenest attacked, at least in America, in the proportion of about 3 : 1; yet this relative difference does not apply so well to early life, nor to adults in other countries. The

¹ Hist. Nat. y. Med. de el Princip. de Asturias, 1762, 327.

² Trans. Nat. Assoc. for the Study of Pellagra, 1912, 18.

³ Amer. Journ. Insanity, 1864, XXI, 223.

⁴ Trans. Nat. Assoc. for the Study of Pellagra, 1912, 23.

⁵ Amer. Journ. Dis. Child., 1912, IV, 172.

⁶ Amer. Journ. Dis. Child., 1914, VII, 124.

⁷ Transac. Nat. Assoc. for the Study of Pellagra, 1912, 333.

⁸ Trans. Nat. Assoc. for the Study of Pellagra, 1912, 39.

disease is very much more frequent in rural districts, and especially under unsanitary conditions and in warm climates. It makes its appearance much oftenest in the spring and summer months. A debilitated state of health seems to predispose to its development. It is not considered communicable, yet a familial occurrence is very frequently observed. Knight,¹ for instance, reported 10 pellagrins in one family, and marked familial predisposition is not uncommon in Italy.

The direct cause is still unknown. The employment of corn-meal as a food had long been considered the chief etiological factor, but later this theory was modified to the belief that the meal must be of an inferior quality. Lombroso² especially has championed the theory, advanced by others before him, that pellagra depends upon the action of a mould infecting spoiled corn, which produces a food intoxication in those eating it. The apparent fact that pellagra occurs also in those who have not eaten corn, and in infants who have been fed only at the mother's breast, as well as the various experimental investigations made, have led to serious doubt of the existence of any etiological relationship between pellagra and corn, or mould growing in it. Alessandrini and Scala³ maintained that the disease is due to the action of silicon in a colloidal form present in the drinking water. Sambon⁴ believed the disease to be an infectious one, dependent upon a protozoon transmitted by the bite of one or more species of *Simulium* fly (black fly; sand-fly; buffalo gnat). Roberts⁵ maintained that it is transmitted by the mosquito. A careful study of the relationship of various insects to the disease made by Jennings and King⁶ led to the conclusion that the *Stomoxys calcitrans* (stable fly) was the probable carrier of the infection. That it may be due to a virus or an organism of very minute size is indicated by the experiments of Harris⁷ who claims to have produced the disorder in a monkey by inoculation with a filtrate from human tissue passed through a porcelain filter. Inoculation experiments by others have been unsuccessful and confirmation is needed.

More recently the prevailing opinion has veered again to the etiological relationship of the food-supply, and the evidence for this is so strong that the disease may be with reason placed provisionally among the nutritional disorders. Goldberger⁸ concluded as a result of dietetic experiments that the cause is a lack of something in the food. This may be a deficiency of protein combined with an excess of carbohydrate, since the introduction of fresh animal and vegetable protein into the diet will effect a cure. In 209 cases in children in two orphanages, 172, seen a year later had, with one exception, under a change in the diet, failed to exhibit any return of symptoms. Wood's⁹ conclusions, based upon his experiments with corn supported the view expressed by others that the cause is a deficiency in the vitamins.

Pathological Anatomy.—The lesions are subject to much variation and are uncharacteristic. Those of the skin are at first erythema; but finally those characteristic of normal old age, such as atrophy, pigmenta-

¹ Journ. Amer. Med. Assoc., 1912, LVIII, 1940.

² Trattato profilattico e clinico della pellagra, 1892. German by Kurella 1898.

³ Contributo nuovo alla etiologia e patogenesi della pellagra, 1914.

⁴ Journ. Tropical Med. and Hyg., 1910, XIII, 271.

⁵ Amer. Journ. Med. Sci., 1913, CXLVI, 233.

⁶ Amer. Journ. Med. Sci., 1913, CXLVI, 411.

⁷ Journ. Amer. Med. Assoc., 1913, LX, 1948.

⁸ Journ. Amer. Med. Assoc., 1916, LXVI, 471.

⁹ Journ. Amer. Med. Assoc., 1916, LXVI, 1447.

tion and sclerotic alterations. Pigmentation may take place in the viscera; the kidney may be cirrhotic; the liver and spleen are often smaller than normal; brown atrophy of the cardiac muscle is common; the fat of the body is much diminished and the muscles atrophied; the fragility of the bones is increased. There may be evidences of chronic inflammatory changes in the brain, meninges and spinal cord. The chief lesions in the nervous system appear to be in the spinal cord, especially the pyramidal tracts, but the posterior columns are also involved. It is questionable, however, whether many of the post-mortem alterations are directly connected with the disease itself, or are not rather those which would be seen in senility or in chronic cachectic states from any cause.

Symptoms and Course.—The method of onset and the symptoms seen are subject to considerable variation. The latter can be divided into *cutaneous*; *digestive*; and *nervous*. Any one of these groups may



FIG. 224.—PELLAGRA.

Case in a girl of 9 years, in the Children's Medical Ward of the Hospital of the University of Pennsylvania, under the care of Dr. M. B. Hartzell. The child had never been outside of Pennsylvania. Courtesy of Dr. Hartzell and of the J. B. Lippincott Co.

appear first or be most prominent. The *cutaneous* are the characteristic manifestations. These may come on very insidiously or suddenly, the first change being a symmetrically developed erythema on the exposed parts of the body, such as the backs of the hands, the face and the neck; or in other cases, if the patient does not wear shoes, also on the feet and legs up to the knees. It resembles sunburn and in mild cases, and especially in young children, may easily escape recognition. In typical instances the erythema becomes darker red and livid, and is followed in about 2 weeks or more by drying, scaling (*dry-type*) and pigmentation of the epidermis (Fig. 224). At the wrists or on the forearms it is sharply demarcated from the healthy skin above it, giving sometimes the appearance of a glove ("pellagrous glove"); or this may be seen on the foot and leg also ("pellagrous boot"), or around the neck ("Casal's necklace"); in fact wherever the covered joins the exposed skin. In some cases vesicles and bulke develop (*wet-type*), or there may be supuration beneath the scaly, crusted epidermis. After a few months in the milder cases recovery occurs and the skin resumes its normal appearance.

With or preceding the cutaneous symptoms there are *digestive* disturbances, generally most marked in children under 4 years of age. Of

these severe diarrhea, often dysenteric, is one of the most prominent and troublesome, and frequently the first to appear. Stomatitis, with redness of the tongue and gums, is often present and is characteristic; salivation may occur, and vomiting is common; or there may be loss of appetite and abdominal pain. *Nervous* and general symptoms, too, may develop at any period. The child may be very little ill, or there may be malaise, loss of flesh, vertigo, depression, insomnia, headache, and cramps in various parts of the body. Occasionally severer nervous symptoms indicating lesions of the cord may be among the earliest manifestations, here especially being paresthesia and, very often, exaggerated reflexes. The examination of the blood reveals nothing characteristic. There may be secondary anemia, but no leucocytosis or eosinophilia.

This is perhaps the history of the first attack. Entire recovery may seem to have taken place; but in the course of the year, sometimes in the autumn but much oftener by the next spring, a second attack occurs, and this recurrence may be repeated during several years as spring comes on, the same symptoms developing but of greater severity and of longer continuance. Sometimes several years may pass by without an attack. Other regions of the body may become involved, as the entire forearms, the shoulders and the genital region; and very occasionally the eruption is universal. The skin in these later attacks is often not so vividly red as in the first, but is more deeply infiltrated and undergoes fissuring and finally atrophy, assuming a permanently pigmented, parchment-like character, with exacerbations of the erythema appearing in the spring-time. The nervous symptoms are more marked in the later attacks, consisting then of very great lassitude, diffuse pains, paralytic or convulsive conditions, mental depression or insanity. Mental disturbances, however, although frequent in advanced cases in adults, are comparatively uncommon in children; except for a degree of dullness or irritability. In the final stage there is profound prostration, with great emaciation and the development of the typhoid state.

Prognosis.—On the whole this is very unfavorable. The death-rate in America according to Lavinder¹ equalled 37 per cent. in the 15,870 cases collected by him. Recovery in fully developed cases is very unusual. Death took place in the 1st year of the disease in 12 out of 55 cases collected by Tucker.² Although apparent recovery often occurs readily, recurrence of the symptoms is very liable to take place; and after perhaps many years the patient dies. Sambon³ reported a case in a woman of 88 years who had the disease since she was 17 years of age. Such a duration as this is, however, unusual. Severe cases may be fatal in 2 to 3 years, but generally the disorder lasts a longer time. There is reason to believe that, although pellagra is more rapidly fatal in infancy, in children from the age of 4 to 10 years the course is milder and recovery may take place under suitable treatment. If the deficiency-theory is correct, there is reason to hope that the prognosis in the future may be greatly better under proper dietetic management.

Diagnosis.—The positive diagnosis can be made only upon the development of the characteristic cutaneous eruption, alone or in combination with the digestive and nervous disturbances described. In regions where pellagra is endemic, at least a provisional diagnosis may be made before cutaneous symptoms are seen, based upon the combination

¹ *Loc. cit.*

² Journ. Amer. Med. Assoc., 1911, LVI, 246.

³ Trans. Nat. Assoc. for the Study of Pellagra, 1912, 87.

of redness of the tongue, diarrhea, lassitude, headache, paresthesia and other nervous symptoms.

Treatment.—Infants with pellagrous mothers should not nurse from them. This is not because the malady is transmissible, but because the milk is generally inferior in quality, and infants continued at the breast develop intestinal disorders and an atrophic state. In cases of any age the most favorable hygienic surroundings should be chosen, including, if possible, change of environment, especially to a cooler climate. Hydrotherapy has been very valuable. The diet, too, should be made abundant and nourishing, and changed in some way from that which has been employed; especial care being taken to diminish the amount of carbohydrate and increase that of animal or vegetable protein. Tonic remedies to improve the general health may be needed, including iron and arsenic either by the mouth or hypodermically; and a search for and removal of associated intestinal parasites is of importance. This treatment is to be followed not only in the developed disease, but as a prophylactic measure, in view of the decided predisposing influence which poverty and ill health exert. For the local treatment exposure to sunshine should be avoided, as this tends to produce or increase the eruption, and soothing applications should be used, such as are indicated in various forms of erythema.

SECTION IV

DISEASES OF THE DIGESTIVE SYSTEM

CHAPTER I

DISEASES OF THE MOUTH, LIPS, JAWS, TONGUE AND SALIVARY GLANDS

DISEASES AND MALFORMATIONS OF THE JAWS

The failure of union of the two sides of the upper jaw is described under **cleft palate** (p. 670); and the narrowing caused by the presence of adenoids is referred to elsewhere (p. 688). **Prognathism** of the upper jaw is not infrequent. It may be a fault of development, or may sometimes be produced by thumb-sucking (Vol. II, p. 284). As a result the upper teeth slant forward beyond the lower. Prognathism of the lower jaw is also encountered. Sometimes the lower jaw is abnormally small (**micrognathia**), the teeth failing to come into position below the upper ones when the mouth is closed. Very occasionally **exostoses** are encountered; or **odontomata** from irregularity in the development of one or more teeth of the second set within the body of the jaws. **Malignant new growths** connected with the jaw are of very great infrequency in early life, the most common being sarcoma. Tumors arising at the junction of the gums with the jaw (**epulis**) are occasionally seen. The

nature of these varies. Generally the growth is a simple granuloma connected with a diseased tooth; but even if sarcomatous, their removal is generally not followed by any return. Maxillary cysts of congenital origin may be seen. **Necrosis** of a portion of the jaw may follow alveolar abscess, ulcerative stomatitis, or noma; and in cases of the last-mentioned disorder in which life is not lost, very great deformity may remain. **Ankylosis** of the jaw may result from noma or from traumatism. A **chronic ostitis** may be produced by tuberculosis or syphilis.



FIG. 225.—HARELIP ON LEFT SIDE.
(Willard, *Surgery of Childhood*, 38,
Fig. 22.)

DISEASES OF THE LIPS

Harelip.—This very common deformity is caused by the failure of the frontonasal plate to unite with the lateral process on one side, thus producing a fissure beneath the nostril (single hare lip). When occurring on both sides double harelip results (Fig. 225). The fissure varies in degree from a slight notching to a complete cleft passing into the nostril. In the latter event, particularly if on both sides, cleft palate also is commonly present.

The fissure may be great enough to interfere with nursing, with consequent inanition from lack of nourishment. Apart from great care

for cleanliness of the mouth the treatment is entirely surgical. Operation should be done at from 2 to 3 months of age unless difficulty in feeding the child makes earlier interference necessary. The infant should be in as good general condition as possible at the time.

Erosions at the Angles of the Mouth (Fäule Ecken; Perlèche).—This condition, long known, but first carefully studied by Lemaistre¹ in 1886 and called La Perlèche, is not of very frequent occurrence. The French name is derived from the tendency of the patient to lick the affected areas (pour lécher).

Etiology.—It effects especially early childhood, being rarely seen in adults; is favored by the presence of a state of debility and by uncleanness; and would even appear to be contagious, since several members of one family may be attacked, or it may occur epidemically in schools. A streptococcus has been described by Lemaistre.



FIG. 226.—EROSIONS AT THE ANGLES OF THE MOUTH.
(Epstein, *Jahrb. f. Kinderheilk.*, 1900, LI, 317.)

Symptoms.—The lesions consist of small, fissure-like ulcers arranged radially at the labial angle, generally at both sides. The skin is red and swollen, and under the influence of the moistening by the constant application of the tongue a grayish ulcer of larger size may develop (Fig. 226). The lesions are painful, but do not affect the child in other respects. The course is favorable and recovery takes place in from 2 to 4 weeks under treatment, leaving no scars in the majority of instances; but without treatment the course may be prolonged and may become chronic. Except in severe cases there is no lymphatic involvement.

Diagnosis.—This is a matter of importance since the lesions bear a resemblance to the plaques of syphilis. They differ in that the fissures never extend into the mouth nor deeply into the tissues; are without induration at the base; and are not strictly limited to the commissures.

Treatment.—In the way of prophylaxis care merely must be taken for cleanliness of the mouth and against possible infection from other children. The fissures should be touched with a solution of nitrate of silver, 10 per

¹ Etude sur l'air de la ville Limoges, de la perlèche, du streptococcus plicatilis. Ref., Comby, *Traité des mal. de l'enf.*, 1901, II, 13.

cent., a crystal of sulphate of copper, tincture of iodine, or burnt alum, and then covered with a healing ointment, as of zinc or bismuth, or have applied compound tincture of benzoin, ointment of yellow oxide of mercury, or 25 per cent. ichthyol.

Other Affections of the Lips, such as syphilitic lesions, herpes, eczema, and the like, are discussed in the sections dealing with these.

ANOMALIES OF DENTITION

Natal Teeth (*Dentitio præcox*).—Very rarely infants are born with one or more teeth. Ballentyne¹ was able to collect but 70 reported cases, and states that in the Paris Maternity from 1858 to 1868 the anomaly occurred but 3 times in 17,578 births. The tendency is in rare instances hereditary. These natal teeth are nearly always the central incisors in the lower jaw. The tooth is usually poorly developed, and often so loosely attached that extraction is necessary. If this is not the case it should be allowed to remain unless it renders nursing too painful to be tolerated. Natal teeth may sometimes be supernumerary and then will be replaced later by the teeth of the first dentition.

Early Dentition.—Dentition may occur remarkably early, and the age of 3 months may see the first incisors appearing. This is an occasional characteristic of hereditary syphilis, but is not a proof of the existence of this disease.

Delayed Dentition.—Much more frequent than prematurity of dentition is that of delay. Within normal limits the first teeth may not appear until the age of a year, but a period longer than this, or even not exceeding it, is a strong indication of the presence of rickets, which is the most frequent cause. Cretinism, too, is attended by delay in the first appearance of teeth.

Irregularities of Dentition.—Disturbed *sequence* in the eruption of the teeth seems to have but little significance. The appearance of the upper incisors before the lower has been claimed (Jacobi),² to be an attendant upon idiocy with premature ossification of the cranium. It certainly, however, occurs in many normal infants. Rachitis is a not infrequent cause of a disarrangement of the sequence. Irregularities in the *shape* and *character* of the teeth are seen especially in the disposition to caries following severe constitutional diseases. This is especially true of rickets and of tuberculosis and the so-called scrofulosis. Furrowing and erosions of the teeth, especially those of the permanent set, may be due to defective formation of the enamel, often dating from the occurrence of stomatitis or of some constitutional illness in early childhood; or the condition may be hereditary. The notching and the development of a peg-shaped form of the permanent incisors in inherited syphilis is also to be noted. (See Syphilis, p. 576.) Other irregularities are sometimes seen, such as that of *size*, certain teeth being abnormally small, or some abnormally large. The *number* of the teeth is sometimes abnormal, one or more teeth being wanting, or occasionally the number being greater than normal. This is more often seen in the permanent set than in the temporary. Failure of the temporary teeth to fall out at the proper time is liable to occasion *malposition* of those of the second set. I have exceptionally seen so many of the first set remaining in position that the child actually appeared to have two rows of teeth in each jaw. Excessive thumb-sucking sometimes forces the incisors of the lower jaw inward and those of the upper

¹ Edinb. Med. Journ., 1896, II, 1025.

² Intestinal Diseases of Infancy, 1887, 102.

jaw outward (see Thumb-sucking, Vol. II, p. 282), as may also the constant sucking at a rubber "comforter." Lip-sucking, too, may cause depression of the lower incisors.

Difficult Dentition.—Formerly it was the general practice of physicians to attribute to "teething" ailments of many sorts which are now generally admitted to have no actual connection with it. Unfortunately the custom is still widespread among the laity, and even to too great an extent in the medical profession as well. Opposed to this view is the more modern one that teething is a purely physiological process and never produces unpleasant symptoms. The truth probably lies somewhere between these extremes, although, with many others, I am convinced that the rôle played by teething in the production of symptoms is a very small one, and that, as a rule, dentition produces only teeth. The error lies largely in assuming that the redness of the gum over a tooth about to appear is an indication of trouble produced by the tooth. The question hinges upon what is to be called the eruption of a tooth. As a matter of fact there is scarcely a time in the first 2 years of life when a tooth is not either pushing through the gums or, more deeply situated, through the alveolar process. If pain and other disturbances are caused by the tooth they should rather occur during the more difficult process—the advance of the tooth through the resistant bony structure—for the thin layer of the gum overlying a tooth which is nearly erupted is, as is well known, comparatively insensitive. Lancing of the gum often relieves a catarrhal stomatitis and the accompanying local and nervous symptoms; but it is the stomatitis, not the presence of the tooth, which is causing the disturbance in the majority of cases, and which is relieved by the operation. This is shown by the fact that the employment of other remedies for the relief of the stomatitis is often promptly followed by disappearance of the symptoms which had been attributed to teething.

Nevertheless there are sometimes instances seen in particularly susceptible children where the close approach of a tooth to the gum and the pressure upon this, without any discoverable stomatitis, produces symptoms promptly relieved by lancing. I have occasionally seen such instances, but I have oftener failed to see any benefit following lancing. Among these symptoms may be mentioned moderate fever, restlessness, disturbed sleep, fretfulness, loss of appetite, diarrhea, and a constant tendency to put the hands into the mouth. Convulsions may very rarely occur, but I have never seen an instance of this which could be unequivocally attributed to teething. As all the symptoms involved may be due to a catarrhal stomatitis, associated with gastrointestinal disorders, or may be dependent upon more remote causes, the necessity of most careful examination of the whole body becomes evident. Lancing of the gums should be the last thought entertained by the physician; not the first, as is too often the case. Moreover, gum-lancing, if not followed by a prompt eruption of the tooth, may readily produce a cicatrix which would increase the supposed difficulty in the eruption which the procedure was intended to alleviate.

Alveolar Abscess (*Gum-boil*).—This is a common affection, especially in those whose teeth have been neglected. Following a pericementitis an abscess develops about the root of a tooth. Pain and swelling result, sometimes with fever. The whole side of the face may be much swollen. The pus usually discharges itself into the mouth, but occasionally externally through the cheek or into the antrum. Treatment consists in the employment of hot applications within the mouth, and the opening of the abscess as soon as possible.

Caries of the Teeth.—This condition should be mentioned especially on account of its deleterious influence upon the system in general. It is of extremely common occurrence even in early childhood. Caries results chiefly from lack of care of the teeth. There is also a predisposition to it in children suffering from depressed health from any source, and it is particularly frequent in rickets. An improper diet is also an active factor, especially the frequent eating of sugar in any form, since the organisms producing the disorder thrive in a sugar-medium. Apart from the direct effect of the caries, such as toothache, imperfect mastication, foul breath, alveolar abscess, forms of stomatitis, and the like, absorption may readily take place through the resulting gingivitis. Tuberculosis of the glands below the jaw and finally even of more distant parts of the body may occur; and it is likely that anemia, continued fever, and acute and chronic rheumatic conditions may be brought about in the same way. In any event it is certain that the treatment of the inflammation of the gums associated with caries appears to be an essential in obtaining improvement of the general condition in many instances. It is probable, too, that the irritation produced by carious teeth is responsible for many nervous conditions, such as habit-spasm in which the face is involved, as well as headache. The importance of effort for the prevention of dental caries is evident and of prompt treatment if already present; and this applies to the first set of teeth as well as the later ones. Many mothers regard it as a matter of indifference whether or not the primary teeth decay, and fail to consult a dentist regarding this. Not only are the results mentioned liable to happen, but it is of frequent occurrence that the 6-year-old molars, erupting as they do without the displacement of any of the primary teeth, come in and decay without this being discovered.

CATARRHAL STOMATITIS

Etiology.—This disorder is not only a very common affection, but is an attendant upon various other diseases. It is oftenest seen in early childhood and especially in infancy. The period of life at which it is most likely to occur is that for the eruption of the temporary teeth. This is not evidence, however, that dentition is in any way causative of it. (See p. 651.) Traumatism of various sorts is active in its production. Here may be mentioned rough washing of the mouth by the nurse, which is a very frequent agent, and the action of hot or chemically irritating substances. A common etiological factor is lack of cleanliness, the result either of neglect or of the introduction of unclean objects into the mouth, and the consequent irritation through bacteriological action which follows. Diseases of the gastroenteric tract are very commonly attended by catarrhal stomatitis, as are many of the acute infectious disorders, especially measles, diphtheria and scarlet fever; and other varieties of stomatitis are constantly accompanied by inflammation of the catarrhal form.

Symptoms.—The mucous membrane of the mouth becomes red, swollen, hot and tender. The change is seen especially on the gums and the tongue, but frequently also on the lining of the lips, cheeks and palate. Slight bleeding may occur. The central portion of the dorsum of the tongue is usually coated, while the tip and edges exhibit swollen and red filiform papillæ. Not infrequently the whole of the dorsum is red. The swelling is especially marked on the mucous membrane about the teeth. The tongue and lips may be fissured. Salivation is decided and often

so excessive, particularly in nurslings, that the secretion overflows the lips, irritates the surrounding skin and moistens the clothing. That the inflammation is painful is evidenced by the complaints of older children, and by the continued restlessness, fretfulness, crying and sleeplessness of infants, and their refusal to take food, although clearly hungry. An attempt to nurse is promptly followed by dropping of the nipple with a cry of pain. Moderate swelling of the lymphatic glands below the jaw may occur. The temperature is normal or slightly elevated. Diarrhea and vomiting may occur, but rather as complications or as causes than as symptoms of the disease.

The **course** of the disease is a few days or a week in length in children under treatment and otherwise healthy. In those debilitated or suffering from other affections it may be much longer.

Treatment.—Food is best given in a cool form and liquid or soft. If sucking is painful, feeding with a spoon can be employed. Should all food be refused, gavage may be used, but this is rarely necessary. Small pieces of ice may be put frequently into the mouth. The mouth should be kept very clean through oft-repeated washing with cold water; a solution of potassium permanganate (1:8000 or stronger); or a saturated solution of boric acid, to which a little tincture of myrrh may well be added (1:24). All rough cleansing processes must be avoided, and a soft pledget of absorbent cotton moistened with the solution employed. Painting with a weak solution of nitrate of silver (1 to 2 per cent.) may be used in obstinate cases. Internal medication is hardly required.

APHTHOUS STOMATITIS

(Herpetic Stomatitis, Maculo-fibrinous Stomatitis, Aphthæ)

The nature of this affection is not well understood. The title "herpetic" has been employed on the theory that the lesions were the counterpart of herpes upon the lips; but this relationship, although possible, has not been proven, and the title "herpetic stomatitis" may well designate inflammation of another variety. (See Simple Ulceration, p. 663.) "Aphthæ" is a title long in vogue and formerly applied to a variety of affections of the mouth.

Etiology.—Age has a predisposing influence, the disease being most common in infancy and early childhood, and especially in the 2d year of life, although not infrequent in adults. In 587 cases in early life studied by Monti¹ the ages were: Under 6 months, 10; in the 1st year, 69; 1 to 2 years, 290; 2 to 3 years, 106; 3 to 4 years, 45; 4 to 14 years, 77. The previous health has no influence except that the disease is not infrequently associated with disturbances of the digestion. Uncleanliness of the mouth appears to be an important predisposing factor. In certain subjects there is seen a decided tendency to frequent recurrence. No specific microorganism has been demonstrated, nor has an infectious quality been certainly proven, although often believed to exist. It has been claimed by Ollivier² and others that the malady is identical with the foot-and-mouth-disease of cattle and transmitted from them, but for this view there appears to be no sufficient basis.

Pathological Anatomy.—The lesions consist of small patches of irregular shape, varying in size from that of a small pinhead to that of a split-pea. They are discrete, or occasionally somewhat confluent. The

¹ Henoch's Festschrift, 1890, 461.

² Rev. mens. des mal. des enf., 1892, X, 11.

lesion appears at first as an elevation of the epithelium with a red margin and a whitish centre. It is produced by cellular proliferation and fibrinous exudation beneath the epithelium, surrounded by a zone of hyperemia. It is very possible that a vesicle first develops but is unrecognized on account of the influence of the moisture of the mouth. In about 24 hours the epithelial covering is lost and an opaque whitish or yellowish patch remains. This is superficial and is level with the surrounding mucous membrane; that is to say, it is not an ulcer in a strict sense, but an infiltration; and if effort be made to remove the exudate a bleeding surface remains. The exudate is absorbed in a few days without necrotic change taking place, and new epithelium spreads over the erosion which remains.

Symptoms.—The principle symptom consists in the presence of the lesions and the local conditions produced by them. The plaques appear in successive crops, their favorite seat being the inner surface of the lips and the edge of the tongue, although any part of the oral cavity may be attacked. The process may occasionally extend to the pharynx and tonsils. The lesions may be few in number and are usually chiefly discrete, but sometimes numerous and coalesce into large, irregular patches. They are painful and tender to the touch, and the taking of food is often almost refused on account of the suffering produced; the pain being similar to that present in catarrhal stomatitis, but greater. The secretion of saliva is increased; the tongue is coated; there may be an odor to the breath, but not of the offensive character present in ulcerative stomatitis.

The general symptoms consist of malaise, loss of appetite, often moderate fever, fretfulness and disturbed sleep; the degree of these corresponding to that of the involvement. The cervical lymphatic glands are sometimes enlarged in severe cases.

Complications.—Catarrhal stomatitis always accompanies the process; herpes labialis is sometimes seen; and vomiting and diarrhea may occur.

Course and Prognosis.—In the milder discrete cases the duration is seldom more than a few days or a week and the prognosis is entirely good. The confluent cases last somewhat longer. If previous constitutional debility exists the duration may be extended to 2 or 3 weeks through the persistent development of fresh crops, and the general nutrition may be affected.

Diagnosis.—As a rule this is unattended by difficulty. The confluent form may suggest diphtheria, especially if the apthous lesions develop first or chiefly in the pharynx; but scattered aphthæ elsewhere in the mouth will settle the question. Ulcerative stomatitis appears chiefly at the junction of the teeth with the gums, and the breath is characteristically offensive. There is also in this a tendency to hemorrhage and to breaking down of the mucous membrane.

Treatment. Prophylaxis.—This is limited to the keeping of the mouth clean and the general health good; yet with our ignorance of the exact cause, preventive measures are unsatisfactory.

Treatment of the Attack.—Thorough gentle washing should be employed freely, as in catarrhal stomatitis, with a cold solution of boric acid (10 per cent.), or of permanganate of potash (1 : 10,000 or stronger). If the healing is tardy the lesions may be touched carefully with burnt alum or nitrate of silver; or painting the lining of the mouth with a 1 per cent. solution of nitrate of silver may be employed. The food given should be soft and unirritating and preferably cool.

THRUSH

(Sprue)

The disease has been known since early times, but its nature not understood until revealed by the studies of Berg¹ in 1841 and of Müller² in 1842. Its frequency has been much diminished as a result of the greater care for cleanliness now exercised.

Etiology.—The affection is seen almost solely at an early age, especially in the first 2 or 3 months of life, although it may occur later in marantic or neglected subjects, especially in institutions for infants; and it may even develop in vigorous infants in perfect health. It can appear, too, in adult life in the course of debilitating diseases. Its occurrence in early infancy is favored by the greater dryness and the greater degree of rest of the mouth and tongue at this period. The presence of gastro-intestinal disorders is a powerful predisposing factor, as is catarrhal stomatitis. Slight injury to the mouth made by roughness in cleansing it also produces the unhealthy local condition necessary for the development of the parasite. The absence of cleanliness acts in a similar manner. The nature of the diet has little influence, since breast-fed children may also be attacked.

That the malady is *infectious* has been shown beyond question. It can certainly be acquired from unclean rubber nipples, or even from the maternal nipple. The germ consists of a mould-fungus of the class of hyphomycetes, but its exact position is even yet undetermined and it has been variously named. It was first described by Berg.³ Robin⁴ named it the *oïdium albicans* and Reess⁵ the *saccharomyces albicans*, while Plaut⁶ identified it with the *monilia candida*. Under the microscope can be seen long, fine, mycelial threads with numerous gonidia, although the organism is subject to considerable variation. There are mingled with these epithelial cells, red and white blood-corpuscles and bacteria. The fungus is present in many normal mouths without producing an infection; may be found readily in healthy stools; and exists to a certain extent free in the air. The germ is, therefore, widespread and only awaits favorable conditions for its development. Outside of the body it grows readily in a solution of sugar or one of gelatine.

Pathological Anatomy.—The parasite enters between the epithelial cells and develops best beneath the superficial layer. Thence spores and mycelial threads penetrate the different lower layers of the epithelium, but generally do not extend beyond this. The process is not productive of pus. The infection may extend to the tonsils and pharynx and rarely to the nasopharynx, middle ear, esophagus, stomach, intestines, larynx, trachea, lungs and skin. Even the walls of the blood-vessels and the tissues may be penetrated in some cases (Heller)⁷ and the disease may exceptionally be spread by the blood, metastasis occurring in remote parts of the body, as the spleen and kidneys (Schmorl)⁸ or the brain (Zenker).⁹ A general infection by thrush has been reported by Heubner.¹⁰

¹ Journ. f. Kinderkr., 1847, IX, 194.

² Arch. f. Anat. u. Physiol., 1842, 193.

³ Loc. cit.

⁴ Hist. natur. des végétaux parasit., Paris, 1853, 488.

⁵ Sitzungsber. d. med.-phys. Societät; Erlangen, 1876 7, IX, 190.

⁶ Kolle and Wassermann, Handb. d. path. Microörg., 1903, I, 575.

⁷ 62 Versamml. deutsch. Naturforsch. u. Aerzte, 1889, 312.

⁸ Centralb. f. Bact. u. Parasitenk., 1890, VII, 329.

⁹ Jahresb. d. Dresdener Gesellschaft. f. Natur. u. Heilk., 1861 2, 51.

¹⁰ Deutsche med. Wochenschr., 1903, XXIX, 581.

Symptoms.—The characteristic symptom is the presence of the parasite in the mouth. Small white patches develop of pin-point size and larger, resembling curdled milk. They are situated chiefly on the dorsum of the tongue, but the lining of the cheeks, lips, and palate is also a favorite locality. They may be few in number and discrete, but oftener are very numerous and tend to coalesce, forming large plaques. The patches are covered with epithelium and somewhat raised, and can be removed only by the exercise of considerable force, a raw, slightly bleeding surface remaining. The surrounding mucous membrane is reddened. As the lesions grow older they become more yellowish and are more easily removable, or loosen of themselves. The mouth is rather dry, and the reaction of the saliva is generally acid; this latter being probably the result of the growth of the parasite rather than a favoring cause, since the fungus thrives best on alkaline or neutral media. Nursing is painful and food often refused; and swallowing, too, causes pain if the disease extends to the fauces. Gastrointestinal disturbance, although often the predisposing cause, may sometimes be a result of the disease; diarrhea being a frequent symptom and producing great irritation of the buttocks. Vomiting may occur and there may be moderate fever. Any marantic condition previously existing is increased by the interference with the taking of food.

Course and Prognosis.—Under suitable treatment in comparatively healthy infants, the duration is short, lasting only 1 or 2 days, and the prognosis is excellent. In debilitated infants, on the other hand, the disease may be obstinate, either entirely unyielding or continually recurring after apparent improvement. In such cases thrush adds to the debility and hastens the fatal ending. The prognosis is also less favorable when the process spreads to the pharynx. In rare instances death has been caused by occlusion of the esophagus.

Diagnosis.—Thrush can scarcely be confounded with any other disease, although superficial examination may cause it to be mistaken for curdled milk. The latter, however, is easily removable and consequently recognized without difficulty. Patches in the pharynx might suggest diphtheria, but the presence of the fungus in the mouth as well excludes this malady. Microscopic examination readily removes all uncertainty.

Treatment.—**Prophylaxis** is most important, and is readily attained by cleanliness of the mouth and of everything which goes into it, including rubber-nipples and the maternal breast. Systematic cleansing must be done with the greatest gentleness in order to prevent the development of abrasions; absorbent cotton wrapped about the finger or on an applicator being employed rather than coarser material.

The **treatment of the attack** itself consists likewise of careful, very gentle, frequent cleansing of the mouth. The patches may be cautiously rubbed away with pledgets of cotton moistened with an alkaline solution, as of borax (10 per cent.), or bicarbonate of soda (6 per cent.), and the mouth bathed 3 or 4 times a day with some remedy destructive to the fungus. Among these substances are permanganate of potash (1:5000 or stronger), liquor formaldehydi (1 or 2:100), or nitrate of silver (1 per cent.). The old-time borax-and-honey should be avoided, since any saccharine substance favors the growth of the fungus. In its place a 25 per cent. solution of borax in glycerin may be used. Such constitutional treatment should be employed as the other symptoms present demand.

ULCERATIVE STOMATITIS

This form of inflammation of the mouth is one of the severer varieties, much less common than those previously described. It has been recognized as a distinct affection only since the last part of the 18th century.

Etiology.—Age is an important factor, the disease being most frequent at from 4 to 8 years and being seldom seen before this period. It appears not to develop until the teeth have erupted. Imperfect hygiene, uncleanness of the mouth, and any debilitating influence strongly predispose. The disease is consequently much more common among the poor than among those of better social condition. It is a not infrequent symptom of scurvy, and may be an attendant upon diphtheria, measles, scarlet fever, typhoid fever, and other infectious diseases. The presence of carious teeth is a predisposing cause, and it may be produced by metallic poisons, especially mercury. It would seem that the malady does not often attack those in previously good general health and with the mucous membrane of the mouth in a normal condition. It often appears in epidemic form in hospital wards, indicating that it depends upon some specific transmissible germ. According to the investigations of Bernheim and Pospischill¹ a fusiform bacillus and a spirochete identical with those seen in ulcero-membranous angina (p. 682) were found in all but 2 of 30 cases of ulcerative stomatitis. The bacillus is long and narrow, larger than the diphtheria bacillus, and with pointed ends (bacillus fusiformis). Two are often joined end to end. This germ is always accompanied by a spirochete much longer and thinner than the bacillus.

Pathological Anatomy.—The lesions usually begin on the free border of the gums. There first occurs hyperemia with swelling from inflammatory exudate; the gum becoming loosened from the teeth, much swollen, bleeding easily, and exhibiting a reddish-purple color. Ulceration now appears upon the free border, indicated by a narrow, yellowish line, due to the development of necrosis in the superficial layer of the mucous membrane. The process rapidly advances in breadth as well as in depth, and commonly spreads by contiguity to the mucous membrane of the cheek and lip. The ulcers which are produced are covered by a grayish or yellowish material, which is firmly adherent to the subjacent tissue. As recovery takes place the necrotic tissue loosens and a new epithelial covering forms. In the most severe cases the necrosis extends into the alveolar process of the jaw, the roots of the teeth may be exposed, and the teeth themselves fall out.

Symptoms.—The character of the local process has already been described. The original lesion in most cases is situated on the external aspect of the gums, usually next to the incisor teeth, sometimes the canines or molars, and oftenest in the lower jaw upon one side only. In most instances the disease spreads to the mucous membrane of the cheek or lip in contact with the original lesion. The edges of the tongue are not infrequently involved. The process may extend to the tonsils and occasionally the palate, or may sometimes originate there. (See Ulcero-membranous Tonsillitis, p. 682.) The first symptoms noticed are usually salivation and a very offensive odor of the breath. The lesions are painful and tender and food is refused, partly from this reason and partly from loss of appetite. The tongue is coated, swollen and often tooth-marked. The ulcerated area bleeds easily if disturbed by examination.

¹ Jahrb. f. Kinderheilk., 1898, XLVI, 134.

Constitutional symptoms may be absent, or in severer cases there may be fretfulness, fever, pallor, swelling and tenderness of the submaxillary lymphatic glands, and decided debility, or even symptoms of a septic nature.

Complications.—Vomiting and diarrhea are sometimes seen, perhaps the result of swallowing the secretion of the ulcers. More or less catarrhal stomatitis is common and aphthous stomatitis may likewise occur. Noma may be a sequel.

Course and Prognosis.—In average cases under proper treatment the attack lasts 7 to 10 days, the slough separates, hemorrhage and pain cease, the salivation and the odor disappear, and recovery is complete. If untreated, however, the disease may continue sometimes for months. In the severer cases, too, where the periosteum becomes involved, the course is more prolonged. Even the milder cases occasionally become somewhat chronic in spite of treatment. Relapses are prone to occur. In the exceptional instances which end fatally, death is due to exhaustion or to some complicating cause, such as sepsis or noma.

Diagnosis.—This is unattended by difficulty in most cases. The situation of the lesion and the offensive odor are characteristic. Aphthous ulcers may in some cases first appear upon the free borders of the gums and, if confluent, may at first cause uncertainty. The patches, however, are superficial and other discrete lesions in the mouth, together with the absence of the odor, make the diagnosis clear. Diphtheria of the mouth is uncommon, nearly always secondary to that of the fauces, and presents really no close resemblance. Noma is characterized by the extensive induration usually in the cheek, and by the black, gangrenous tissues. I have known severe ulcerative stomatitis with involvement of the alveoli and loss of teeth to be designated noma; but the process in the latter condition is much more severe. The diagnosis of the cause of stomatitis should also be made when possible, particularly with reference to the existence of some constitutional condition, the influence of mercury, or the presence of scurvy.

Treatment. Prophylaxis.—This consists in the removal of all predisposing causes by improving the general health, attending to cleanliness of the mouth, the treatment of carious teeth, and, in hospital practise, the isolation of those already attacked. Particular caution must be observed after the occurrence of acute infectious diseases.

Treatment of the Attack.—The cause must be sought for and removed if possible, and measures must be taken to improve the general health, to guard against relapses by the administration of tonic treatment, often with iron, and especially to see that sufficient food is taken, even by gavage if necessary. The food should naturally be of an unirritating, cool, and easily digestible nature. For local treatment frequent washing of the mouth with a solution of permanganate of potash (1:1000) or with peroxide of hydrogen (10 to 50 per cent.) should be used. A solution of nitrate of silver (5 to 10 grains (0.32 to 0.65):1 ounce (30), is sometimes serviceable in obstinate cases. The best and almost a specific treatment for the disease is, however, the administration of chlorate of potash. As this is promptly excreted by the saliva, it may be given internally in doses of 3 grains (0.19) every 2 hours for a child of 4 years. It should be well diluted, as its contact with the lesions causes some pain. The possible deleterious effects of the drug must be borne in mind. All carious teeth or portions of necrosed bone must be removed and a stronger solution of nitrate of silver or other caustic applied carefully to the diseased locality.

GANGRENOUS STOMATITIS

(Noma. Cancrum Oris)

The term "noma" is of a wider application than "gangrenous stomatitis" since a condition of the same nature as that occurring in the mouth is occasionally seen in other parts of the body, as the ear, nose, genitals, or anus, and is properly designated by this term. The description which follows applies to the mouth only. The disease appears to have been more common in earlier times and at present is fortunately rare, inasmuch as it is one of the most dreadful of the disorders of early life. Woronichin¹ reported but 22 cases occurring among 8286 sick children in hospital-practice, and Ranke² gave even a lower incidence of 2 in from 4000 to 5000 patients.

Etiology.—Age is an important predisposing factor, the disease being oftenest seen between 2 and 5 years of age. There is no certain proof of any contagiousness, although there is a great possibility that this exists. Local epidemics of noma in infant's homes or hospitals have been described, as by Blumer and MacFarlane³ and by Neuhoof,⁴ not all the cases, however, being limited to the mouth. It is less frequent in infants, although cases are recorded in the early months of life. The 46 cases reported by Woronichin,⁵ including those seen both in hospital and dispensary practice, were from 1 to 2 years, 8; 2 to 3 years, 12; 3 to 4 years, 6; 4 to 5 years, 7; 5 to 6 years, 4; 6 to 7 years, 6; 7 years, 1; 8 years, 1; 12 years, 1. Previous bad health is almost a necessity. The affection is met with oftener after typhoid fever and especially after measles than under any other conditions, although it may follow pertussis, scarlet fever, diphtheria, enteritis, tuberculosis or other affection occasioning general debility. In the same way residence in damp, unhealthy localities, or the presence of ulcerative stomatitis, may act as a predisposing cause. That the affection is dependent upon the action of some germ seems beyond question, but the nature of this is not certainly known, and it is very probable that more than one microorganism is capable of producing it, or a symbiosis of a number of organisms. Various pus-producing germs have been found in the gangrenous tissue. Certain microorganisms have been reported as specific. Schimmelbusch⁶ described a bacillus, sometimes short and sometimes in long threads; Ranke⁷ found cocci; and growths of the leptothrix type have been observed. A bacillus identical with, or closely resembling, the diphtheria-germ has been seen in a number of instances, as in 2 case of my own reported by Sailer.⁸ Hellesen,⁹ observed a diplococcus, and Durante¹⁰ a staphylococcus. A number of observers have noted the fusio-spirillary germs characteristic of ulcerative stomatitis and ulcero-membranous angina. These were seen in 1 of my cases which began in the tonsil.

Pathological Anatomy.—The process is a rapidly spreading gangrene with the characteristics of this condition wherever found. There is little disposition to limitation shown, except in the favorable

¹ *Jahrb. f. Kinderheilk.*, 1887, XXVI, 161.

² *Jahrb. f. Kinderheilk.*, 1888, XXVII, 309.

³ *Amer. Journ. Med. Sci.*, 1901, CXXII, 527.

⁴ *Amer. Journ. Med. Sci.*, 1910, CXXXIX, 705.

⁵ *Loc. cit.*

⁶ *Deutsche med. Wochenschr.*, 1889, XV, 516

⁷ *Loc. cit.*

⁸ *Amer. Journ. Med. Sci.*, 1902, CXXIII, 59.

⁹ *Jahrb. f. Kinderheilk.*, 1908, LXXVII, 294.

¹⁰ *La Pediatria*, 1902, X, 232.

but rare cases where a line of demarcation forms. It is probable that the mucous membrane is first attacked, although sometimes the process would appear to have started in the substance of the cheek; and very exceptionally the lesion is first seen on the cutaneous surface.

Symptoms.—The commonest situation of the disease is the cheek opposite the molar teeth and upon one side only, rarely both sides being involved simultaneously. Often, however, the gum or the inner surface of the lips is the primary seat. Examination of the oral cavity reveals a small ulcer covered by a dark-grey slough. This spreads in area and in depth, and produces a black, gangrenous area, with the surrounding tissue edematous and swollen. Meanwhile induration develops in the



FIG. 227.—GANGRENOUS STOMATITIS BEGINNING IN THE CHEEK.

Child of 7 years, admitted to the Children's Hospital of Philadelphia, suffering apparently from typhoid fever. Gangrenous process began Dec. 10, with swelling of the right cheek; Dec. 19, almost entire cheek involved; Dec. 23, lower lip and chin attacked; Dec. 24, death.

substance of the cheek, which, seen from without, is much swollen, shining, pale, and hard to the touch. Soon the skin over it becomes red, and then blackish (Fig. 227) and the cheek perforates. The process follows a similar course when the gums are affected. Wherever the primary seat, the destructive process spreads with astonishing rapidity; involving the alveolar process; causing the teeth to loosen and fall; attacking often the tongue, palate, lips, or a large portion of one side of the face; and finally, in the worst cases, extending to the other side (Figs. 228, 229). Often the first symptom noticed is the excessively offensive odor of the breath, and as the case progresses this becomes intense and very penetrating. The general condition of the patient is but little affected at first, but as the disease advances debility becomes



FIG. 228.—GANGRENOUS STOMATITIS BEGINNING IN THE LIP.

Same case as in Fig. 230. Photograph taken soon after admission to the Children's Hospital Oct. 25.



FIG. 228.—GANGRENOUS STOMATITIS BEGINNING IN THE LIP.
Same case as in Fig. 228. Photograph taken a few days later.

great, the pulse is feeble, delirium and insomnia may occur, diarrhea develop, and fever of an uncharacteristic type be present (Fig. 230), and the symptoms are those of sepsis. Pain is but little marked. The sub-maxillary lymphatic glands may be enlarged.

Course and Prognosis.—The course of the disease is generally very rapid, visible changes taking place almost from hour to hour. Perforation may sometimes occur in 24 hours, but oftener not before 3 or 4 days. The usual duration is 5 to 10 days. Sometimes, however, the course is much slower and the induration may last several days before gangrenous changes appear. In cases which recover a line of demarcation forms and the slough is thrown off after 8 to 10 days, sometimes

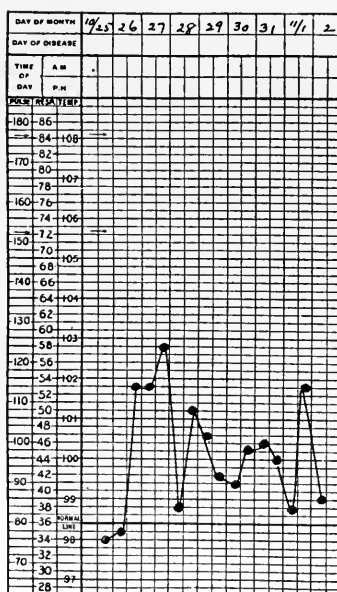


FIG. 230.—GANGRENOUS STOMATITIS.

Lena P., aged 2 years, admitted to the Children's Hospital of Philadelphia, Oct. 25, with sloughing in the right half of the upper lip; Oct. 28, gangrene has extended rapidly, and now involves all the region of the mouth (Fig. 229), extending within it and perforating the left cheek; thorough cauterization on this date; Nov. 2, died.

without perforation occurring, although this is exceptional. The great majority of cases die either from exhaustion or from a complicating pneumonia. At the best 75 per cent. of them end fatally; and when recovery does occur it is nearly always with extensive deformity remaining. The more widespread the lesion and the poorer the general health of the patient, the more serious is the outlook.

Complications.—Bronchopneumonia is common; abscess or gangrene of the lung sometimes occurs; severe hemorrhage is rare. Diarrhea is so frequent that it is to be regarded rather as a symptom of the disease.

Relapse and recurrence have been very occasionally seen.

Diagnosis.—A mistake in diagnosis can hardly be made. Ulcerative stomatitis does not produce a gangrenous slough even when severe and causing necrosis of the jaw, and does not involve the skin. It is only

when noma complicates a previously existing ulcerative stomatitis that it is difficult to determine the time of onset of the former.

Treatment. Prophylaxis.—The maintenance of the general health and the careful attention to the mouth in the course of infectious diseases or in any form of stomatitis is of the greatest importance. Other children should be separated from a patient with noma.

Treatment of the Attack.—Prompt employment of surgical interference by thorough excision, with subsequent cauterization, offers most hope of arresting the disease. The cauterization is best done by heat or nitric acid, carbolic acid, or a paste of chloride of zinc. Nicoll¹ reports a case of recovery following the intravenous injection of salvarsan. Exposure to rays from a red electric lamp was successful in the hands of Motschan.²

SIMPLE UCLERATION OF THE MOUTH

(Herpetic Stomatitis, etc.)

By this term may be designated the small ulcers, usually single or few in number, which frequently develop as a result of catarrhal stomatitis or from other causes, including digestive disturbances. In exceptional cases the lesions may be very numerous and widespread over the oral cavity, as in a case reported by Scheltema.³ Some of the cases are probably closely allied to herpes. The infectious diseases, such as measles, typhoid fever, and the like, may sometimes produce the condition, and the eruption of varicella in the mouth might be classed here. The process differs from true ulcerative stomatitis in the situation and the usual smallness in size and extent of the lesions. The ulcers are unlike those of aphthous stomatitis in that they exhibit a shallow depression with superficial necrosis rather than the firm, elevated mass of the latter disease. They probably begin in many cases as vesicles, but the covering mucous membrane promptly ruptures. They are benefited by cleanliness and the application of a 1 per cent. solution of nitrate of silver.

SECONDARY STOMATITIS

Here may be included certain forms not already mentioned, which are secondary manifestations to other diseases:

Gonorrheal Stomatitis.—This has been reported as affecting the new born, the infection taking place from the maternal passage during birth (Rosinski).⁴ It is also acquired in other ways, as by self-infection from a gonorrheal vulvovaginitis. A whitish deposit appears in the mouth, especially upon the palate and the tongue and the gonococcus is discoverable on microscopic examination. Recovery takes place in a few days. Cleanliness is the only treatment required, or perhaps the application of a 1 per cent. solution of nitrate of silver.

Diphtheritic Stomatitis.—This form of stomatitis is a rare and dangerous development, the pseudomembrane appearing on the lining of the cheeks, lips, or tongue. It occurs only in the worst cases of diphtheria. A faucial involvement nearly always precedes it. In one instance I found the lesion in the mouth before discoverable in the pharynx. The pseudo-membrane has the usual appearance of diphtheria of other

¹ Archives of Pediatrics, 1911, XXVIII, 912.

² Arch. f. Kinderheilk., 1904-5, XL, 241.

³ Med. Maandscr. v. Verlosk., Vrouwenz. en Kindergeneesk., III, 524. Ref., Jahrb. f. Kinderh., 1915, LXXXI, 546.

⁴ Deutsch. med. Wochenschr., 1891, XVII, 569

localities. It is to be distinguished only from extensive confluent aphthous stomatitis, and this is usually easily done if attention be paid to other symptoms.

A pseudomembranous stomatitis, other than diphtheritic, may be the result of trauma, especially from the action of hot or caustic fluids in the mouth, or be produced by other germs, as the pneumococcus, perhaps in the course of pneumonia; or by the staphylococcus or streptococcus in connection with sepsis elsewhere. The constitutional symptoms are severe.

Syphilitic stomatitis has already been considered in discussing syphilis. It shows itself in the form of mucous plaques and in fissures at the labial commissures, or sometimes in acquired cases in the form of the primary lesion within the buccal cavity.

BEDNAR'S APHTHÆ

(Pterygoid Ulcer)

This condition first described by Bednar¹ is in no way related to aphthous stomatitis, and the name is in so far confusing. Strictly speaking, it is an affection of the palate.

Etiology.—It is seen only in infants in the early months of life and is, in reality, a trauma produced by too forcible cleansing of the mouth, or by the pressure of too long a rubber nipple. The mere act of sucking by a child with catarrhal stomatitis may cause it, the mucous membrane of the palate being tightly stretched and anemic and consequently very easily injured.

Symptoms.—These consist of the local lesion, which is a superficial ulcer situated on the posterior portion of the hard palate in the median line, or upon one or the other side of it. Sometimes there are two ulcers symmetrically placed, one on each side of the raphe. The ulcer is shallow, one-quarter to one-half inch in diameter and covered by a greyish or yellowish-white exudation. It is painful and causes difficulty in sucking, with consequent progressive loss of weight and strength. Catarrhal stomatitis, thrush, or gastrointestinal disturbance may occur as a complication.

Course and Prognosis.—The course of the disease is usually favorable, the ulcer healing in a few days, or in 1 or 2 weeks. In cases in infants with great malnutrition it may prove very obstinate and the bone may become involved. The lesion may extend and a pseudodiphtheritic membrane or ulcerative condition develop, and the infant die of sepsis or debility.

Treatment.—This consists, first of all, in preventing the development of the lesion by the avoidance of any possible mechanical irritation. A similar caution will usually permit of rapid healing of an ulcer already present. In more obstinate cases very careful, frequent washing with a solution of boric acid may be employed, with occasional application of one of nitrate of silver (1 per cent.). A weak solution of cocaine (1 per cent.) may be carefully applied before nursing if pain is great, but much caution is required against constitutional effects.

DISEASES OF THE TONGUE

Apart from the disorders of the tongue seen in some affections of the mouth, and in those nervous diseases which involve the organ, several conditions deserve consideration here:—

Macroglossia.—This is an anomaly rarely seen as an affection existing alone and of congenital origin. In such cases it is in reality a diffuse

¹ Die Krankheit. d. Neugeb. u. Säugl., 1850, I. 105.

lymphangioma, and may reach such size that the tongue cannot be retained in the mouth and interferes with nursing and later with speech. Enlargement of another nature, not congenital and of a less degree, is seen in cretinism, mongolian idiocy and acromegaly. The lesion here is a hypertrophy of the muscles and of the interstitial connective tissue. The treatment of congenital macroglossia is surgical, if the size interferes with the taking of food. If it depends upon cretinism, the thyroid treatment suitable for this disease is indicated.

Very rarely other congenital defects of the tongue are encountered, among them congenital **absence** of a portion of the organ, or in other cases a split or **bifid** tongue.

Tumors.—These are not of frequent occurrence. Among them may be mentioned dermoid cysts, diffuse or more localized angiomas and lymphangiomata, and growths consisting of misplaced thyroid tissue. Surgical treatment is indicated.

Tongue-tie.—By this title is designated a shortening of the frænum linguæ, making it impossible to protrude the tongue beyond the teeth to the normal extent. It varies considerably in degree. As a rule it gives no trouble, but in well-developed cases may interfere with sucking and later with perfect articulation. Such a condition is, however, certainly most uncommon. Treatment consists in nicking the edge of the frenum with blunt-pointed scissors and in tearing through the remaining membrane. In this way the occasional danger of hemorrhage is averted, except in bleeders.

Ulceration of the Frenum.—This is often seen in pertussis, or in other conditions attended by violent coughing. It is the result of the forcible impact of the frenum against the lower incisor teeth. It occasionally, however, appears in other conditions also, or without discoverable cause in nursing infants. The application of a weak solution of nitrate of silver soon produces a cure.

Sublingual Fibroma (*Sublingual Granuloma; Riga's Disease; Fede's Disease*).—This affection appears to be of not infrequent occurrence in Italy, and a few cases have been reported in Poland, France, Germany, Austria, and elsewhere. A case occurring in America was described by Amberg¹ in an infant of American descent. The first description to attract attention was by Riga.² Why the disease should be seen especially in Italy is not yet explainable.

Etiology and Pathology.—The disorder is seen only in the 1st year of life, and especially the latter portion of this, after the lower central incisor teeth have erupted. It results from irritation produced in the action of sucking, through the friction of the neighborhood of the frenum of the tongue against the teeth, or against the hard ridge of gum if the teeth have not appeared. An inflammation results, with hyperplasia of the tissues, and an indurated, small, tumor-like body of the nature of a papilloma is produced by the fibrinous infiltration. Later a superficial ulcer may form in cases of long duration, and a superficial small-celled infiltration results with the characteristics of a granuloma. The development of the disease is not, as a rule, influenced by any previous disturbance of health. Other disorders may accompany it but bear no etiological relationship, although they may influence the duration of the process.

Symptoms.—There are generally none other than the sublingual growth. The earliest appearance is that of an indurated, greyish,

¹ Amer. Journ. Med. Sci., 1903, CXXVI, 257.

² Movimento medico-chirurgico, 1881, XIII, 22. Ref. Amberg.

opaque, hard, somewhat flattened elevation, which increases rapidly in size until it reaches perhaps one-quarter to 1 inch in diameter, situated transversely at the position of the frenum (Fig. 231). The central region is grey, and the remaining portion redder in color. The **duration** of the lesion may be weeks or months, and the **prognosis** is entirely favorable. In subjects with debilitating diseases it may last much longer, but will finally disappear, unless the complicating affection is the cause of death.

The **diagnosis** is to be made only from sublingual ulceration, such as is seen often in pertussis. In sublingual fibroma, however, the process is hyperplastic, and any ulceration present is superficial.



FIG. 231.—SUBLINGUAL FIBROMA.

(Amberg, *Amer. Jour. Med. Sci.*, 1903, CXXVI, 257.)

Treatment.—This consists chiefly in the employment of boric acid washes and the application of tincture of iodine. In obstinate cases it may be necessary to remove the central incisor teeth. In debilitated subjects every measure should be employed to improve the general health.

Black Tongue (*Nigrities Linguae*).—Black tongue is not a common affection, Brosin¹ having been able to collect the published reports of only about 40 instances. An interesting case in a child of 2 years is illustrated by Gottheil.² Its cause is not known with certainty. The etiological action of a bacterium or of a mould has been invoked, as has the existence of a hyperplasia of the epithelial layer of the filiform papillae, with subsequent hardening and change of color by pigmentation. The lesion appears in the form of irregular areas situated chiefly near the base of the tongue, or running forward in a long streak toward the tip. The black area may be either smooth or of very decidedly hairy appearance if the papillae are unusually long. To these latter cases the title "*black hairy tongue*" has been given. There are no subjective symptoms of

¹ Die schwartze Haarzunge, 1888.

² Arch. of Pediat., 1899, XVI, 255.

importance; seldom more than a sense of dryness. The disease sometimes disappears quickly, but is oftener very chronic, lasting perhaps for years. In the line of treatment, mouth washes of borax, hyposulphite of soda, or hydrogen dioxide may soften the papillæ and remove the color; or, if these are unsuccessful, applications of weak solutions of nitrate of silver or of salicylic acid may be used. Cure may be permanent, but oftener the discoloration returns.

Glossitis.—This is uncommon in early life except as a result of injury of some sort. The edges of the tongue may be irritated by carious teeth, or the dorsum by corrosive or hot fluids. The stings of insects are an occasional cause. Rarely urticaria may involve the tongue. The inflamed portion is painful and deep-red, with prominent papillæ. In severe cases the whole tongue may be badly swollen and there is salivation, moderate fever, and difficulty in swallowing and in speaking. The swelling may be so considerable that the tongue protrudes beyond the lips, and even breathing may be interfered with. The condition is not usually serious. The course is slow, the swelling subsiding in a few days and leaving superficial ulceration in the severer cases. Very rarely a parenchymatous inflammation of the tongue occurs, with the formation of an abscess in the body of the organ. The treatment of glossitis consists in purgation, frequently repeated introduction of ice into the mouth, and, in bad cases, scarification. The food should be liquid, given through a nasal tube if necessary.

Tongue-swallowing.—This rare condition is occasionally seen in infants who are suffering from nasal obstruction. Attention was first called to it by Bouchut.¹ The efforts of the child to breathe cause a drawing backward of the body and tip of the tongue until they are pressed against the hard palate, cutting off more or less completely the entrance of air. Treatment consists in drawing the tongue forward and maintaining it in this position. At the same time efforts must be made to open up the nasal passages.

Geographical Tongue.—Epithelial desquamation of the tongue, wandering rash of the tongue, lichenoid of the tongue, ringworm of the tongue, and various other titles have been applied to this common disorder, many of them showing our lack of complete knowledge of its nature.

Etiology.—The cause is uncertain. Although oftenest seen in childhood, it is frequent, too, in infancy and sometimes continues into adult life. It has been supposed to be syphilitic, scrofulous, or parasitic, but without good reason. It certainly often occurs in subjects suffering from chronic intestinal indigestion or the exudative diathesis, but by no means only in these. Hygienic conditions appear to have no positive influence.

Symptoms.—The lesion begins as a small, greyish-white spot on the surface of the tongue. The patch enlarges rapidly and irregularly, while simultaneously the white color disappears from the central portion, leaving a bright-red hue, due to the loss of epithelium and of the filiform papillæ. The slightly raised grey border, consisting of epithelial thickening, advances rapidly over different parts of the tongue, the outline changing daily. Meanwhile, although the region immediately within the border is still bright-red and denuded of epithelium, the central, older portion of the lesion gradually assumes the normal smooth, glistening appearance of the healthy tongue, through the development of new epithelium upon it. The irregular border gives to the condition the title "geograph-

¹ *Maladies des nouveau-nés*, 1885, 8th Ed., 279.

ical tongue" (Fig. 232) from the similarity to the outline of a map, while the term "wandering rash" indicates the characteristic constantly changing appearance and position. Several patches are usually present at one time, often partially fusing into each other, and new ones may form as the older disappear, and perhaps spread again over the regions first attacked.

There are no subjective symptoms produced by the lesions, and the disease is entirely harmless. The individual patches persist about a week, but the course as a whole is essentially chronic, lasting sometimes months or years. No treatment is needed, nor is any effective.

DISEASES OF THE SALIVARY GLANDS

Malformations of the salivary glands are of very uncommon occurrence, among them being a defective formation and a congenital salivary fistula. **Tumors** are rarely encountered. **Concretions** may rarely



FIG. 233.—BILATERAL SECONDARY SUPPURATIVE PAROTITIS AFTER TYPHOID FEVER.

Case in a boy of 11 years, in the Children's Ward of the University Hospital, Philadelphia.

be found in any of the salivary ducts, or, less often, in the glands themselves. Neuhof¹ could collect but 7 instances of the disease in children, to which he added 3 others. A primary purulent **sialoadenitis** of the submaxillary or sublingual glands is occasionally seen in the first weeks or months of life (Mikulicz and Kümmell).² The parotids are not involved. There is swelling of the glands, febrile symptoms develop, and pus is discharged through the ducts. Often distinct abscess-formation occurs. The prognosis is usually good.

Ranula.—The title is employed to designate a cystic formation in the mucous glands or the sublingual salivary glands. It is only rarely

¹ Amer. Journ. Dis. Child., 1916, XI, 232.

² Die Krankh. d. Mundes, 1898, 228.

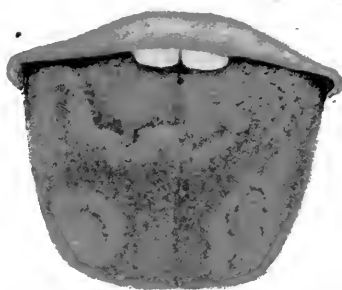


FIG. 232.—GEOGRAPHICAL TONGUE.

Showing the denuded areas with elevated, thickened margins.

found in early life, but may even be congenital, and 8 such cases were collected by Le Guay.¹ If of considerable size, the tongue is pressed upward and may interfere with sucking.

Secondary Parotitis.—The primary form of parotitis has already been considered. (See Mumps, p. 494.) Secondary parotitis is sometimes seen even in early infancy. It may occur after stomatitis or purulent otitis; or in a worse form, usually advancing to suppuration, it may complicate severe fevers, especially typhoid fever and septic infection (Fig. 233). The germs enter through the duct of Steno, or by way of the blood vessels in metastatic cases. The process is generally unilateral. The *symptoms* are the usual ones of inflammation, combined with local pain, swelling, and tenderness. After several days resolution begins in favorable cases; but not infrequently there is deep-seated suppuration with increase of swelling and tenderness, discoloration appears, and finally pus is discharged through the overlying skin, or into the mouth either through the duct or the tissues of the face. Necrosis of the jaw or severe hemorrhage may occur; or sometimes death take place from sepsis. In the line of *treatment* cold compresses should be applied and a purgative given. If resolution does not promptly occur, surgical aid should be sought before fluctuation develops.

Mikulicz's Disease.—This is a very uncommon affection of which Tileston² could find but 12 reported cases occurring in children. It was first described by Mikulicz³ in 1892, and consisted, according to him, of a bilateral, painless enlargement of the salivary and lachrymal glands. Some cases reported after his publications have been associated with changes in the blood suggesting pseudoleukemia or leukemia, and with decided enlargement of the lymph-nodes and spleen. These cases should probably be excluded. There may, however, be a moderate enlargement of the lymph-nodes and of the spleen present. The *etiology* is obscure. Mikulicz considered the condition the result of the action of some one or several unknown infectious agents. Tuberculosis would seem to have been the cause in some instances. The histological changes are those of lymphoma or of chronic inflammation. There are practically no *symptoms* except the bilateral enlargement mentioned, and perhaps a disturbance of the functions of the glands, resulting in dryness of the mouth and the absence of tears. Evidences of inflammation are wanting. The *course* is slow, but the *prognosis* fairly good in cases uncomplicated by serious changes in the blood. In the line of *treatment* arsenic or the iodides may be given. The employment of the Röntgen-ray or excision may be needed in cases not yielding to medical treatment.

Salivation.—An unusually free flow of saliva, or at least an inability to retain it in the mouth, is seen in infants of 3 or 4 months at the time the secretion is normally fully established. After this period increased flow may depend upon a variety of causes, especially stomatitis of any form and particularly that produced by the administration of mercury; or may be caused by reflex influences, such as nausea or gastralgia; or may be a symptom of certain nervous affections. The duration and prognosis depend upon the cause, and treatment is to be directed to this.

¹ Thèse de Paris, 1911.

² Trans. Amer. Ped. Soc., 1911, XXIII, 356.

³ Beiträge z. Chirurgie, Billroth's Festschrift, 1892, 610.

CHAPTER II

DISEASES OF THE PHARYNX AND PALATE

DEFORMITIES AND NEW GROWTHS OF THE PHARYNX AND PALATE

Syphilitic ulceration may take place in the pharynx, oftenest in the palate. **Tuberculosis** of the pharynx, not including the tonsils, is of very infrequent occurrence, and especially so in early life. **Stenosis** may be a congenital deformity, or result from syphilis or trauma. **Morbid growths** are uncommon, those oftenest seen being lymphoma and fibroma. Congenital **perforation** of the palate may occasionally be observed. If developing later, it is oftenest the result of hereditary syphilis. A **high and narrow arch** to the palate may attend the presence of adenoid growths, or may occur independently of this and be the cause of some of the symptoms usually attributed to the vegetations (Landsberger).¹ It sometimes accompanies mental defect.



FIG. 234.—CLEFT PALATE WITH DOUBLE HARELIP.

Courtesy of Dr. H. R. Wharton.

Cleft Palate.—This common condition consists of a congenital fissure of the palate, oftenest of the soft palate only but not infrequently of the bony portion as well, in bad cases extending into a corresponding fissure in the lip. In the worst cases the palate exhibits a double cleft connecting with a double hare-lip, and leaving the inter-maxillary process completely separated from the jaw on each side (Fig. 234). An opening through the palate necessarily interferes with sucking, since the abnormal passage into the nose prevents the formation of the necessary vacuum. As a consequence the nutrition of the infant is severely affected and special methods of feeding are necessary. A long, large, rubber nipple attached to a glass tube and rubber bulb, as in the Breck Feeder for premature infants (p. 256, Fig. 45) is often of service;

¹ Arch. f. Kinderh., 1915, LXV, 113.

or a nipple may be employed to the upper side of which a flap of thin rubber is attached in order to close the gap in the palate when the infant sucks. In other cases feeding by gavage is necessary. In spite of all care severe cases generally die, not so much from lack of food, as on account of the attending constitutional debility usually present. If the child survive, operation is best deferred until the 2d year or later.

Paralysis of the Palate and Uvula.—This is seen especially in diphtheria, but may occasionally develop in the course of other infectious diseases, and in certain disorders of the nervous system.

Bifid Uvula.—The uvula may be split into two portions at its tip, or the division of the two halves may extend throughout the length, producing a double uvula. There are usually no symptoms present.

ACUTE CATARRHAL PHARYNGITIS

(Simple Angina)

Distinction is to be made in etiology between the very frequent acute pharyngitis occurring as a primary affection, and that seen in the course of many of the infectious diseases, especially diphtheria, scarlet fever, rubella, grippe, and often measles, or accompanying some form of stomatitis. Acute catarrhal tonsillitis is to be viewed as often a part of this disease and may be discussed in this connection. The symptoms of primary and of secondary pharyngitis are largely the same, and any differences will be considered with the separate diseases with which secondary pharyngitis is associated. The primary affection alone will be considered here.

Etiology.—Pharyngitis is exceedingly common at all ages, although infants in the 1st year, and especially in the first 6 months of life are probably less predisposed than later. Exposure to cold and wet, dwelling in overheated rooms with subsequent local chilling from drafts, debilitated health, nasopharyngeal obstruction, and similar causes produce a susceptibility, and are certainly common etiological factors. Digestive diseases not infrequently are attended by irritation of the pharynx. There is, too, a remarkable predisposition in certain individuals or families, in some instances dependent upon a tendency to rheumatic manifestations. The action of irritant substances, such as hot or caustic liquids, is an occasional factor. All the conditions mentioned act as predisposing causes, rendering the tissues subject to an invasion by pyogenic germs from the mouth. No one germ, however, appears to be specific.

Symptoms.—The disease begins acutely, often with slight fever and malaise and accompanied by the symptoms of acute rhinitis and laryngitis. All four regions may be involved simultaneously, or not infrequently the pharynx is first attacked and the nose, larynx and trachea later. There is an uncomfortable sensation in the throat, which varies from that of slight roughness or dryness to severe pain especially on swallowing. The inflammation often produces a frequent, annoying cough. In well-marked cases decided fever is present, with chilliness, malaise and sometimes headache. These are most marked when the tonsils are especially involved. The submaxillary lymphatic glands may be somewhat swollen, and there may be stiffness and aching in the neck. In infants and young children the onset is sometimes quite severe, with vomiting, high fever and perhaps convulsions, and the patients look quite ill, although at this age they often cannot or do not make complaint

of trouble in the throat. In fact, it is only in older children that any dependence can be placed upon the statements regarding the sensations in this region. At times a gulping method of swallowing or a refusal to take food may be a suggestive indication. The speech is frequently affected, enunciation being interfered with by the pain which the effort evokes.

The local appearance consists of deep redness and more or less swelling of the pharynx, soft palate, uvula, tonsils and pillars. The mucous membrane is at first dry; later covered with thick, tenacious mucus especially on the posterior pharyngeal wall. All these tissues may be involved or only some of them, and to a varying degree. The mild cases exhibit only a slight, bright-redness; the severe ones a deep-red color with swelling. In some severe cases there is little swelling, but the redness and the pain are very prominent features.

Course and Prognosis.—The course of the attack is usually short, complete recovery, under treatment, occurring in from 3 to 4 days or even sooner. Not infrequently, however, if rhinitis and laryngitis are not present at the onset, these develop later and the process then extends downward to the trachea and bronchi; or the pharyngeal process may be slow in recovery or show a tendency to frequent relapses. The association of follicular tonsillitis with catarrhal pharyngitis is very common.

Diagnosis.—This consists mainly in determining whether the condition is a primary one, or is dependent upon some febrile or other disease. Scarlet fever often exhibits sore throat as its first symptom, and the diagnosis must be reserved until later and catarrhal diphtheria may cause confusion. In both disorders a history of exposure to infection is sometimes of aid, and in the latter a bacteriological examination will settle the question. The pharyngitis of grippe can be distinguished only by the prevalence of other symptoms of the disease in epidemic form.

Treatment.—**Prophylaxis** is important in those especially predisposed. Judicious hardening should be employed through the use of general cool morning bathing or local cold bathing of the throat; life more in the open air; avoidance of hot, close rooms; and similar hygienic measures. For the **treatment of the attacks** a saline purgative may be administered at the beginning, a hot general bath or a mustard foot-bath given, and the child then confined to bed and well covered to induce free perspiration. Older children may in addition be given a bowl of cracked ice with instructions to allow the pieces to melt as far back in the throat as possible. A diaphoretic fever-mixture containing aconite and potassium citrate is of service, or if there is much pain small doses of antipyrine or phenacetin. Gargles of normal saline solution or of tannic acid, such as infusion of *rhus glabra*, are very popular and serviceable remedies in children old enough to use them. Lozenges containing menthol, or an oily spray of the same (menthol gr. 2 (0.13); petrolatum liq. fl.oz. 1 (30)) often give much relief. Other lozenges containing eucalyptus, krameria, or guaiac are serviceable. Lime-drops are often useful and agreeable to the child. Inhalation of watery vapor with benzoin is of value if there is much pain. Cold compresses to the throat are sometimes very useful. In the latter stages, and sometimes even from the onset, painting the throat with glycerine of tannic acid gives great relief and hastens recovery, although in some instances it is irritating. In other cases a 25 per cent. solution of argyrol may be employed with benefit.

CHRONIC PHARYNGITIS

This is a condition less often seen in children than in adults, yet not uncommon. It may follow chronic digestive disorders or repeated attacks of acute pharyngitis, but its more frequent cause is obstruction to respiration through the nose. Involvement of the base of the tongue and of the tonsils may occur as complications. The **symptoms** consist of slight discomfort in the throat and efforts to clear it, together with an irritating cough, sometimes moderate and almost continuous, sometimes severe and occurring paroxysmally and only at certain periods, and brought on by the mucus descending toward the larynx. The cough is often falsely assigned to bronchitis. Inspection of the throat shows enlarged, prominent red or yellowish distended follicles dotted especially upon the posterior wall of the pharynx, with the surrounding mucous membrane more or less inflamed, or of a pale color and often partly covered by mucopurulent secretion. The vessels may be visibly dilated. There is no fever and no actual pain, unless exacerbations occur. Deafness may develop as a complication, but this is dependent rather upon accompanying adenoid growths of the nasopharynx. In advanced cases atrophy of the mucous membrane of the pharynx develops. The course is chronic and the disease yields slowly even under treatment.

Treatment consists in removing any nasal obstruction or affection of the tonsillar tissues; attending to the state of the digestive apparatus; and the avoidance of all irritation by exposure and the like. General tonic remedies and change of air are also often necessary. In addition, local treatment is required consisting in the use of alkaline sprays, if there is much irritation, and the cauterization of the individual follicles if not too numerous.

Uvulitis.—Inflammation of the uvula occurs often as one of the symptoms of acute pharyngitis, although occasionally it is largely localized in the uvula. In some cases it may be the direct result of trauma. There is swelling and redness of the part, generally with edema and pain on swallowing; and if elongation is present and the uvula comes into contact with the base of the tongue there is a harassing, tickling cough. Applications of glycerin of tannic acid, a weak solution of nitrate of silver, or of epinephrine (1:1000) are of advantage, as is the sucking of ice. In bad cases scarification of the uvula may be required.

Edema of the Uvula.—This is a condition frequently attendant upon uvulitis. In addition to this, edema may occur in nephritis and in hydremic states. The local treatment is similar to that required for uvulitis. Remedies directed to any more general cause may also be needed.

Elongated Uvula.—With acute or chronic pharyngitis, or independently of these and perhaps of congenital origin, some degree of elongation of the uvula may be observed. The condition depends upon inflammation in the acute cases, and upon relaxation, or an unusual thickening of the mucous membrane in those following a chronic inflammation. There is no sensation of sore throat experienced unless pharyngitis is also present, the only symptoms being an annoying, tickling cough and a desire to clear the throat, worse especially when lying down at night, and produced by the uvula coming into contact with the base of the tongue. The cough may be so severe that vomiting results. Examination of the fauces discovers the uvula touching the tongue if the breath is held or air is expired through the nose. Treatment consists in measures

for diminishing the size of the uvula. If the condition is due to inflammation or relaxation, the application of astringent solutions, such as glycerine of tannic acid, or of adrenaline, is often promptly efficacious. Astringent lozenges, as of eucalyptus, are also of service. In obstinate cases dependent upon a congenital relaxation or upon chronic thickening amputation of the tip of the uvula may be required, but other treatment should first be given through trial.

PSEUDOMEMBRANOUS PHARYNGITIS

(Septic Sore Throat; Pseudodiphtheria)

Etiology.—These titles are but some of those applied to different groups which appear to constitute varieties of one disorder. Pseudomembranous tonsillitis is included in this description. The disease may be primary, or be secondary to some other affection, especially the acute exanthemata, such as measles and, most of all, scarlet fever; although often seen, too, in typhoid fever. In many of these the disease may closely resemble diphtheria. As a primary affection it may occur without the previous existence of any of the acute infections. Thus in a very severe form accompanied by extensive necrosis it may occasionally be seen in the new born (Epstein),¹ and in these cases it perhaps complicates Bednar's aphthæ produced by injury to the mucous membrane through rough, unskillful washing of the mouth and throat, or may be consecutive to and associated with thrush. In the form frequently designated "*septic sore throat*" it has often appeared epidemically. A number of such instances have been described in Europe, Savage² having collected and reviewed 18 English epidemics. In 1911 to 1913 several large local outbreaks occurred in the United States, notably in Boston and Baltimore (Ruhräh),³ and in parts of New York State (Bigg)⁴ and elsewhere, the largest being an extensive epidemic affecting over 10,000 persons in Chicago (Capps and Miller).⁵ In several such epidemics the disease has been traced directly to the milk supply.

The primary source of the affection in whatever form it manifests itself is undoubtedly microbic. The germ is much most frequently the streptococcus pyogenes, sometimes associated with the staphylococcus aureus or albus. At times the staphylococcus alone is found, and the pneumococcus and other germs are occasionally observed. The influenza bacillus appears to be the agent in some instances; the diphtheria bacillus is never present. The relationship of the pseudodiphtheria bacillus to pseudomembranous inflammation is not yet definitely settled. The epidemic outbreak would indicate that the disease is transmissible both indirectly, as by milk, and perhaps by direct contact.

Pathological Anatomy.—The mucous membrane of the throat is often deep red, and there is prone to develop a pseudomembrane, either on the tonsils alone or more widely spread. In structure it is practically identical with that of diphtheria, but macroscopically it may differ somewhat from this in being more friable, softer, and more easily removable. The process shows the same tendency as does diphtheria to involve the uvula, the pillars, and in fact all the neighboring tissues; and it may go on to extensive necrotic destruction or even to gangrene.

¹ Jahrb. f. Kinderh., 1895, XXXIX, 420.

² Milk and the Public Health, 1912. Ref., Hamburger, Johns Hopk. Hosp. Bull., 1913, XXIV, 1.

³ Amer. Journ. Dis. Child., 1912, IV, 301.

⁴ New York Med. Rec., 1915, LXXXVIII, 945.

⁵ Journ. Amer. Med. Assoc., 1912, LVIII, 1848.

The germs are found in large numbers in the pseudomembrane and in the underlying mucous membrane and neighboring lymphatic glands, and even in the blood. The glands in the neck may be involved, and in bad cases other organs are attacked by the general septic process, especially the kidneys and the lungs. In the milder cases no pseudomembrane develops, or it is superficial and separates easily.

Symptoms.—The degree of local and constitutional involvement varies greatly. In the primary cases of the *epidemic type* (*septic sore throat*) as seen in the United States, the onset is generally abrupt, with chilliness, fever, headache, vague general pains, sometimes nausea and vomiting, and occasionally a convulsion. A diffuse and often dusky redness of the pharynx and pillars appears, and in a few hours patches of exudation usually develop on the tonsils or pharynx, which can, as a rule, be wiped off without difficulty. In 2 or 3 days the glands in the neck become more or less enlarged and tender, but rarely suppurate. Some cases are much more severe and exhibit hyperpyrexia and great glandular swelling; and there are instances seen during epidemics which run a malignant course and are fatal with septic symptoms in a few days. Milder cases may have no pseudomembrane, but exhibit a diffuse, dusky redness of the throat; and in others there is only the appearance of a lacunar tonsillitis.

In primary cases *not epidemic* in type, and in many of the *secondary* cases, the affection is generally milder and is limited chiefly to the tonsils. The attack may first appear as a lacunar tonsillitis, and only later develop more the pseudomembranous form. The deposit can be removed with comparative ease and with little damage to the tissues. The tonsils and pharynx are red and swollen. The onset is usually sudden and often stormy, with high fever, headache, generally pain in the throat especially on swallowing, and sometimes vomiting. The enlargement of the submaxillary lymphatic glands is usually only slight and the prostration much less than in diphtheria. Not infrequently, however, especially in the secondary cases, the attack may be much more severe than this. In this event the membrane may spread very extensively, involving the pharynx and nasopharynx, the nose, and even exceptionally the larynx, exactly as in some cases of diphtheria; the glandular swelling is often very decided; the temperature high; the pulse weak; prostration great, and the child is apathetic, delirious, or in stupor. There is, in fact, the evidence of a toxic state, and hemorrhagic or other cutaneous eruptions may be present, and grave and even fatal septic complications may develop. Some of the cases are probably instances of erysipelas of the throat.

Complications and Sequels.—The mildest cases show practically none. In the severe ones cervical lymphadenitis is not uncommon and may even be suppurative; laryngitis may develop, especially in cases consecutive to measles; rhinitis and otitis are seen chiefly in cases occurring after scarlet fever; bronchitis, septic nephritis and bronchopneumonia are not unusual, and vomiting and diarrhea may occur. Arthritis has been not uncommon in the epidemics reported.

Course and Prognosis.—The prognosis on the whole is favorable. The duration is but 3 or 4 days in the milder cases, and recovery is rapid. In the severer forms the course is longer, lasting perhaps 1 to 2 weeks and the result uncertain, the process extending both into the substance of the tonsils and to other regions. Extensive sloughing sometimes takes place, all the symptoms of sepsis develop, and the case ends fatally. Fortu-

nately this outcome is uncommon in the primary cases, although oftener seen in those secondary to other diseases. Occurring in early infancy in debilitated subjects the disease is usually fatal.

Diagnosis.—The affection causing most difficulty in diagnosis is diphtheria. In average cases of pseudomembranous pharyngitis the onset is more sudden and stormy than in diphtheria, and the deposit is oftener limited to the tonsils and is not so closely adherent. There is less swelling of the cervical glands and less prostration of the system, except in some of the severe cases of epidemic septic sore throat. The extension of membrane beyond the tonsils is a strong evidence that the disease is diphtheria. There are, however, so many exceptions to these distinctions that dependence should be placed only upon bacteriological examination. The milder primary cases without pseudomembrane do not present the same diagnostic difficulties. The secondary cases are usually more easy of recognition, owing to the presence of other diseases such as measles or scarlet fever; but here, too, a positive diagnosis can be made only by bacteriological study.

Treatment.—Cases of this disorder should be isolated as in lacunar tonsillitis. While waiting for the result of bacteriological examination the patient should be given the benefit of the doubt and receive diphtheria-antitoxin. In the severe cases vigorous local treatment may be required, yet not at the expense of the patient's strength. In general, this is similar to that described for anginose scarlatina and consists of syringing or spraying the throat and nose with warm alkaline solutions or normal salt solution; the use of antiseptic and astringent gargles, or the application to the throat of diluted peroxid of hydrogen (1:4) followed by astringents, such as tincture of the chloride of iron (1:4), glycerine of tannic acid, or nitrate of silver (5 to 10 per cent.). An ice-bag should be applied externally and the general strength maintained by tonic and stimulant remedies.

RETROPHARYNGEAL ABSCESS

This disease, although not common as compared with other affections of the pharynx, is seen not infrequently in the practice of those having to do with many sick children. Bókai¹ states that in 438,799 sick children in the Children's Hospital in Budapest in 49 years there were 926 cases of retropharyngeal adenitis or abscess. Retropharyngeal adenitis not advancing to suppuration and without clinical symptoms is probably not at all an uncommon affection.

Etiology.—Age is a factor of great importance, nearly all cases occurring in the first 3 years of life (Bókai), and especially in infants under 1 year. Only when the abscess is secondary to caries of the vertebrae is it more likely to develop after the period of infancy. Catarrhal conditions of the nasopharynx commonly precede the disease and are the starting point. Consequently abscess is seen oftenest in the colder season of the year when catarrhal states are most common. Exceptionally some infectious disorder, as scarlet fever, grippe, or measles, is the predisposing cause. A debilitated condition of health is also a factor of importance, although children in previously good general health may be attacked. The germs found in the abscess are oftenest streptococci.

Pathological Anatomy.—The lesion consists usually of an inflammation developing in the retropharyngeal lymphatic glands. The process is exactly similar to that of lymphadenitis elsewhere. The glands

¹ *Traité des mal. de l'enfance*, Granchev, 1904, II, 70.

may produce a prominent swelling in the pharynx without the production of pus, but oftener suppuration takes place. The swelling is usually localized more upon one side or the other, inasmuch as the glands are situated upon each side of the median line. In the much less common cases in which the process is secondary to caries of the cervical vertebræ, the pus may sometimes burrow downward along the spinal column.

Symptoms.—The first suggestive symptom appearing after the development of fever of undiscovered cause, or perhaps attending a nasopharyngeal catarrh, is difficulty in swallowing, which may become so decided that no food can be taken, and efforts to accomplish it are followed by regurgitation through the mouth or nose. Associated with this

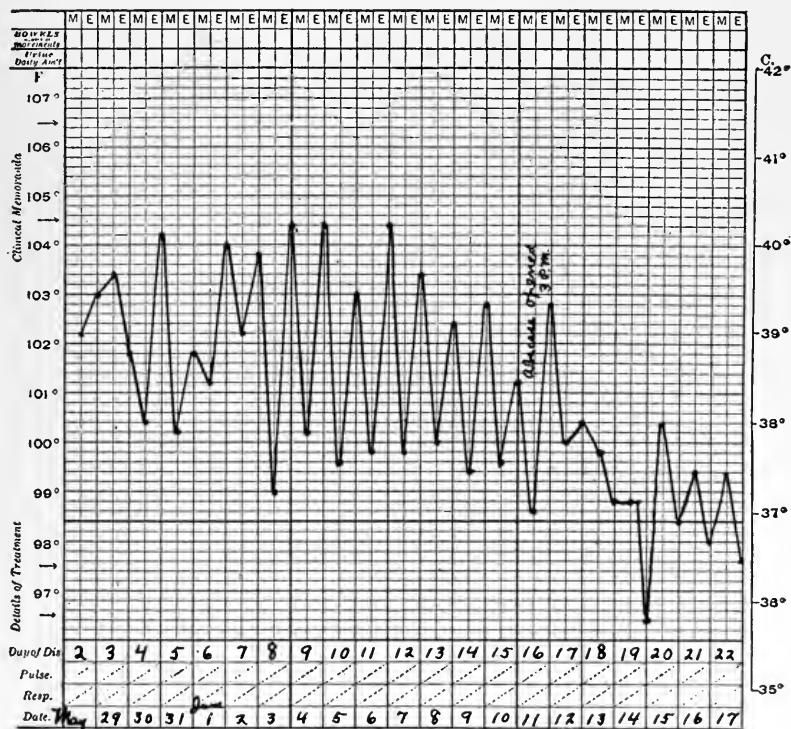


FIG. 235.—TEMPERATURE CHART FROM A CASE OF RETROPHARYNGEAL ABSCESS IN A CHILD OF 20 MONTHS.

(*Hand, Annals of Gynec. and Pediat.*, 1899, XII, Jan.)

is usually an interference with respiration which makes it of a very characteristic nature. It becomes, namely, a spluttering or gurgling, as though there were an accumulation of mucus in the back of the throat; but there is usually not the stridor of laryngeal disease. The mouth is kept open and the head rigidly thrown backward and occasionally to one side. Respiration through the nose is obstructed and there is snoring during sleep. Sometimes in advanced cases there is dyspnea from pressure of the abscess on the larynx. The voice is nasal but usually not hoarse. Slight cough is not uncommon. The constitutional symptoms may be inconsiderable, but debility and more or less fever are the rule (Fig. 235), vomiting and loss of appetite may be present, and sometimes

prostration is decided. In all cases with evident symptoms direct examination of the pharynx reveals sometimes to the eye and always to the palpating finger a fluctuating mass projecting forward from the postero-lateral pharyngeal wall, most marked on one side, and sometimes extending so far forward that the pharynx is nearly filled and the uvula is displaced laterally. The mucous membrane of the pharynx is of a red color. In some cases the swelling is visible from without, upon one side of the neck behind and below the angle of the jaw and extending backward and downward. This may be dependent either upon pus burrowing in this direction, or upon involvement of other neighboring lymphatic glands.

Should the glandular swelling be found early the fluctuating character is absent and the mass is merely indurated (Retropharyngeal adenitis); but usually pus has been formed by the time the characteristic symptoms appear. In abscess in older children, dependent upon caries, the onset is more insidious, the constitutional symptoms generally less marked, and there is little if any fever.

Course and Prognosis.—Occasionally enlargement of the retropharyngeal glands disappears without suppuration. This is uncommon however, in patients who have exhibited clinical manifestations. The course, as a rule, is rapid, and in from a few days to a week the condition becomes apparently a most threatening one. If there is early discovery of it and prompt surgical interference, the prognosis is good in the majority of cases. Sometimes, however, in young infants the constitutional depression is so severe that death occurs even though the abscess has been satisfactorily opened. In other cases the cavity constantly refills and gradual failure of strength takes place. The majority of deaths occur in subjects in whom the diagnosis has not been made. Here sudden death may follow from edema of the glottis or pressure upon the larynx, or may result from rupture of the abscess into the larynx and consequent asphyxia; or, escaping asphyxia, the patient may later die from a septic aspiration-pneumonia. Instances of erosion of blood vessels and fatal hemorrhage are on record. In other cases the pus may burrow outwardly and open on the side of the neck; or, more unfavorably, may pass downward into the mediastinum. The actual death-rate is, however, low; scarcely 4 per cent., according to the statistics of Bókai.¹

When the abscess depends upon cervical caries the course is much slower and operation is prone to be followed by a reaccumulation of pus and sometimes by final exhaustion of the patient. There is also a greater tendency to burrow in different directions, but less danger of asphyxia.

Diagnosis.—This is very misleading to those who have not encountered the disease; easy, however, on digital palpation. This examination is very important. It must be made thoroughly, with the finger guarded against biting by the patient, but rapidly in order not to cause interference with respiration. Apart from the results of palpation the characteristic symptoms are the position of the head, the open mouth, the spluttering respiration and the difficulty in swallowing. *Laryngeal stenosis* may be simulated if the abscess is rather deeply situated and presses against the larynx. The history of the case and the presence of other symptoms as described will generally remove the danger of mistake. *Edema of the glottis* has none of the characteristic symptoms and especially no evidence of abscess found on digital examination. Whether the abscess depends upon lymphadenitis or is secondary to cervical caries can-

¹ Jahrb. f. Kinderh., 1892, XXXIII, 360,

not always be determined. In the latter condition, however, the patients are usually older; the symptoms come on much more slowly with previous ill health; the abscess is more in the median line: and there is more likely to be swelling visible upon the side of the neck externally. There is also greater stiffness of the neck with other symptoms of involvement of the cervical vertebræ.

Treatment.—Surgical intervention should be practised as soon as an abscess is detected. Except in cases of caries, or unless the glandular abscess is already pointing externally in the neck, incision from within the mouth is greatly to be preferred. The child should be seated in an upright position and firmly held with its arms pinioned; a bistoury wrapped with adhesive plaster, except the point, introduced along the guiding finger placed in the mouth; incision made, and the child's head rapidly bent forward and downward to prevent the entrance of pus into the larynx. As there is always some danger of the edges of the clean-cut wound reuniting and the abscess refilling, a good substitute measure is to push blunt-pointed scissors into the abscess and then separate the blades and withdraw them while in this position. Immediate and striking relief follows the operation and in the majority of cases there is no further trouble.

CHAPTER III

DISEASES OF THE TONSILLAR TISSUE

Tumors of the Tonsillar Tissue.—These are of rare occurrence, papilloma and fibroma being those oftenest seen. Lymphosarcoma of the faucial tonsils is also encountered. Tuberculosis of the tonsils and adenoids, of a nature discoverable clinically, is of rare occurrence. Pathologically, tubercle is found in about 5 per cent. of tonsils examined. (See Tuberculosis, p. 557.)

Acute Catarrhal Tonsillitis.—This is a part of *acute catarrhal pharyngitis* and its symptoms do not differ from those already considered under that heading. *Pseudomembranous tonsillitis* has been described in discussing Pseudo-membranous Pharyngitis (p. 674).

ACUTE LACUNAR TONSILLITIS

(Follicular Tonsillitis)

Although quite distinct from catarrhal tonsillitis, the lacunar form shades into it in many cases, one or more small engorged lacunæ often appearing in the catarrhal variety and then disappearing quickly. The more typical cases are here described in which the inflammation is located chiefly in the crypts of the tonsils.

Etiology.—The disease is very common at all ages, except in infancy and especially in the 1st year of life, although it may occur at this period. As with catarrhal tonsillitis exposure to cold and wet, and the occurrence of digestive disturbances are not infrequent causes. There is likewise an undoubted contagiousness sometimes seen. In many children there is a remarkable individual predisposition, and in them attacks are of great frequency. Subjects with a tendency to rheumatism or born of rheumatic parents are very susceptible to the disorder. In fact, inflammation of the tonsils or pharynx must often be considered as one of the manifestations of rheumatism. (See Rheumatism, pp. 622, 626.) The presence of

chronic tonsillar hypertrophy is likewise a powerful etiological factor. Under the influence of such predisposing causes bacteria of various sorts, oftenest staphylococci, pneumococci and streptococci, penetrate into the lacunæ and induce inflammation there.

As a *secondary* affection, lacunar tonsillitis is especially common in the course of scarlet fever and diphtheria, and is considered under these headings.

Pathological Anatomy.—The process consists primarily of an inflammation produced by the action of the invading bacteria. There results an inflammatory swelling of the lymph-follicles of the tonsils, which blocks up the exit of the lacunæ of which the follicles with their epithelial covering form the walls. The proliferated and desquamated epithelial cells, with lymphocytes and bacteria, as well as with the fibrin sometimes produced under the influence of the inflammation, are thus retained in the form of small, whitish or yellowish masses, which finally are discharged as the swelling about the mouths of the crypts diminishes. With this process is combined an inflammatory enlargement of the whole tonsil, and often more or less general pharyngitis. The exudate may disappear very rapidly, and reappear if the opening of the crypt is again closed.

Symptoms.—The attack begins with chilliness, malaise, pain in the head and body, loss of appetite, and fever which quite commonly reaches 103° or 104°F. (39.4° or 40°C.) or over. Vomiting and diarrhea sometimes occur, the tongue is coated, and the breath heavy. The throat is sore and swallowing and speaking painful, though often not to such a degree as in many cases of catarrhal tonsillitis, and small children with the disease may maintain that they have no pain in the throat. Moderate swelling of the cervical lymphatic glands may be present. An erythematous rash may sometimes be observed and lead to an erroneous diagnosis of scarlet fever.

Examination of the throat shows swollen, deep-red tonsils with few or many irregularly shaped yellowish-white spots of varying size. The spots differ in their apparent nearness to the surface, some being well within the crypts of the tonsils and some already discharging. Not infrequently, owing to the close apposition of the lacunæ and the amount of cheesy secretion, the tonsil seems to be covered by a uniform deposit suggesting a diphtheritic pseudomembrane. In such cases, however, the secretion can be more or less completely removed by gentle rubbing with a cotton swab, without any bleeding surface remaining. The process is usually bilateral, although by no means always equally or simultaneously developed on the two sides.

Course and Prognosis.—Although the general and local symptoms are usually severe, they are of short duration, lasting a few days up to a week or occasionally longer. Convalescence is rapid, yet relapses may occur when recovery seems about to begin. The constitutional symptoms are usually over before the local ones have entirely disappeared. The disease has a favorable issue in the vast majority of cases, but not always so, at least as regards complications, since the tonsil while in its inflamed condition permits ready access of germs into the system. Cardiac complications may consequently quickly follow tonsillitis, or rheumatic arthritis develop. Acute nephritis occasionally occurs as a complication, as does septic involvement, especially of the joints.

Diagnosis.—In cases where the deposit has become confluent lacunar tonsillitis may readily be confounded with *diphtheria*. The fact

that the deposit is an easily removable secretion and not a necrotic destruction of the mucous membrane is readily apparent in most cases. In many instances, however, diphtheria begins as a lacunar inflammation or even remains so, and in such only bacteriological examination can settle the question. (See Diphtheria, p. 451.) The presence of membrane upon the pillars or uvula as well as on the tonsils excludes the diagnosis of mere lacunar inflammation. *Scarlet fever* exhibits lacunar tonsillitis often upon the first day of the attack. The presence of other symptoms of this disease determines its nature in most cases. *Aphthous inflammation* may develop upon the tonsil, but has no real resemblance to lacunar tonsillitis and is accompanied by lesions elsewhere in the mouth.

Treatment.—Inasmuch as there is so often a possibility of the tonsillitis being really diphtheritic the patient should be isolated and in doubtful cases a culture should be taken. Even if diphtheria be proven to be absent, continued separation of the patient is wise, owing to the contagious character of simple lacunar tonsillitis sometimes observed. The patient should be at rest in bed and be given small doses of antipyrine or phenacetin to control fever and relieve pain. The diet should be fluid or semisolid. When there is suspicion of the disease being rheumatic in nature, salicylates in some form should be administered. The sucking of small pieces of ice gives great relief, and an ice-bag may be applied over the tonsillar region. For this purpose two small bags of thin rubber, placed one on each side and kept in position, answer much better than the single long sausage-shaped bag often sold for this purpose. This latter is usually much too thick, and does not adapt itself well to the neck. The bandage to maintain the ice-bags should go over the top of the head; not around the neck. In other cases hot applications to the neck give more relief. Cleansing sprays are indicated, such as liq. sod. boratis comp. (Dobell's solution), or hydrogen dioxide. Astringent gargles are of service if the child is old enough to use them properly. Painting the tonsils with glycerin of tannic acid, tincture of the chloride of iron (1 : 3 of glycerine) or solution of nitrate of silver (10 per cent.) is often of value. My preference is for the iron preparation. When such local treatment causes great opposition on the part of the patient, it may be substituted by the giving of tincture of chloride of iron internally in doses of 4 minims (0.25) hourly at 5 years of age. No water should be swallowed immediately afterward, in order that the drug may remain in contact with the tonsils. In constantly recurring cases of lacunar tonsillitis with a tendency to glandular enlargement, removal of the tonsils may be advisable.

CHRONIC LACUNAR TONSILLITIS

This disorder of the tonsils is sometimes a sequel to repeated attacks of acute inflammation. It is also an attendant in many instances upon hypertrophy of the tonsils. There is a retention of the caseous material in some of the crypts. This may be pressed out, or is discharged by the act of coughing, with a resulting fetid odor to the breath. Sometimes the substance cannot be discharged owing to inflammatory adhesions closing the orifice of the crypt. The subjective symptoms are generally limited to attacks of coughing and to slight pain or discomfort attending the swallowing of saliva, although solid food causes no such sensation. At times acute exacerbations occur, with fever developing and with increase of pain. Treatment consists in opening and canterizing the affected crypts; but removal of the tonsils gives a more satisfactory result.

ULCERO-MEMBRANOUS TONSILLITIS

(Plaut-Vincent Angina)

Although the disease was known for some years previously, its dependence upon specific germs was not recognized until the investigations respectively of Plaut and of Vincent. Clinically it closely resembles many cases of pseudomembranous pharyngitis, and only the presence of the characteristic microorganisms can render a distinction possible. The germs producing it are those seen in ulcerative stomatitis (p. 657), the pathological process the same, and the two conditions may be associated. It is of much less frequent occurrence than other forms of inflammation of the tonsils, but by no means uncommon.

Etiology.—As in ulcerative stomatitis, the disease is often epidemic in hospitals or families, indicating that it is of a somewhat contagious nature; yet some predisposition to it must be required, or it would be seen more frequently. It occurs oftenest in children and in subjects in debilitated health. The active cause appears to be a symbiosis of the fusiform bacillus and a spirochete, as already described under Ulcerative Stomatitis. Both these organisms were referred to by Miller¹ as occurring in the mouth; but their association with this form of tonsillitis was first emphasized by Plaut in 1894,² and later by Vincent in 1898.³

Symptoms.—On one or, less often, both tonsils there develops an exudate covering an ulcer of varying depth. In the *milder* cases the deposit can be readily removed, leaving a bleeding surface beneath. A firm pseudomembrane, such as is seen in diphtheria, is not produced. There is little fever and little, if any, constitutional disturbance. An offensive odor of the breath is present. The lymphatic glands at the angle of the jaw are swollen, but do not suppurate. In the *severer* cases the ulceration is much deeper, the constitutional symptoms become more marked and the odor may be extremely offensive.

Course and Prognosis.—In the milder cases the course is short, the exudate soon disappears, and recovery follows in a few days. In the severer the course is tedious, the process may last several weeks, and relapses may occur. The disease may spread to other parts of the pharynx and even prove fatal through the development of extensive necrosis or of gangrene, the child then dying of a condition similar to and probably identical in nature with gangrenous stomatitis. (See p. 659.) I have seen this occur, but such a result is infrequent, and recovery nearly always takes place.

Diagnosis.—The distinction is to be made especially from diphtheria. The softness of the deposit in the milder cases and the greater depth of the ulceration in others are indications against diphtheria; but the only certain test is a bacteriological examination, and even this sometimes permits of doubt in instances where the germs of the two diseases chance to be combined. There are numerous cases on record supposed at first to be diphtheria, but showing only the presence of the fusospirillary organisms. This was true in 73 out of 265 cases suspected of being diphtheria, and reported by Helm.⁴ On the other hand, Reiche⁵ reported 22 typical cases of Vincent's angina with the simultaneous presence of the diphtheria bacillus.

¹ Deutsche med. Wochenschr., 1884, X, 395.

² Deutsche med. Wochenschr., 1894, XX, 920.

³ Bull. soc. des hôp., 1898, XV, 244.

⁴ Journ. Michigan State Med. Soc., 1910, IX, 381.

⁵ Med. Klinik, 1914, No. 33. Ref., Monatsschr. f. Kinderh., Referat., 1915, XIV, 359.

Treatment.—This is similar to that useful in ulcerative stomatitis, chlorate of potash being the best internal remedy, combined with the local application to the tonsils of antiseptic solutions, such as nitrate of silver (gr. 5 to 10 (0.32–0.65) : oz. 1 (30)); tincture of iodine, peroxide of hydrogen (10 to 50 per cent.) and tincture of the chloride of iron (1 : 3 of glycerine). In severe cases supporting treatment is required.

PARENCHYMATOUS TONSILLITIS

(Phlegmonous Tonsillitis, Quinsy, Peritonsillitis.)

Etiology.—Quinsy is an uncommon condition in early life. In infancy it is rare, and only in later childhood oftener seen, even then being much less frequent than in adults. It is generally in reality a peritonsillar abscess, the process often involving the tonsil as well. Less frequently it develops primarily in the tonsil. Catarrhal or lacunar tonsillitis may act as a predisposing cause, and there exists also a decided family or individual tendency. The exciting cause is infection by some pyogenic microörganism.

Symptoms.—The attack begins with suddenness and severity, with the ordinary symptoms of catarrhal pharyngitis. Rapidly, however, the fever becomes high and the pain in the throat constant and severe, making swallowing, speaking, or opening of the mouth difficult or impossible. The breath is offensive; the tongue badly coated. Inspection at first shows little except a prominence in the tonsillar region upon one side of the throat, but palpation reveals a hard, swollen, and very tender mass. The absence of any positive evidence on inspection is due to the fact that the inflammation is largely at first in the deeper portion of the affected region, around or behind the tonsil, and the mucous membrane is not primarily inflamed. As the process advances the whole tonsillar region on one side becomes very prominent, with the mucous membrane red and swollen and the mass pushing the edematous uvula to the other side of the throat and often apparently nearly closing the fauces. The other tonsil and the pharynx in general may exhibit a catarrhal inflammation. Finally fluctuation may sometimes be made out, although in other instances this is never discovered.

With these local manifestations there are increasing symptoms of illness; fever continuing high, oppression and difficult respiration being sometimes present, the pulse rapid, and general restlessness and delirium perhaps occurring. There may be much pain and tenderness on moving the neck. The patient is able to take almost no food or drink and the general condition is most distressing.

The disease lasts several days or a week, and then an abscess may often be seen to be pointing, generally in the neighborhood of the anterior pillar of the fauces. Pus may be discharged and relief from all symptoms be complete in a few hours. In many cases, however, there is no pus evident and a gradual resolution takes place. Severe hemorrhage has been known to follow the bursting of the abscess, and edema of the glottis also has occurred. The **prognosis** is, however, nearly always good.

Diagnosis.—This is usually easy after the first few days. Palpation is often of greater aid than inspection at the beginning of the attack.

Treatment.—Early in the case the effort should be made to abort the process. This may sometimes be done by the application of ice-bags

externally over the affected region, combined with the sucking of pieces of ice. Later the only methods open are the efforts to give relief from pain. The use of ice should be continued. Sometimes the application of a hot poultice externally and the employment of hot water as a gargle give more relief. Opiates internally are often indispensable. As soon as any region suggesting fluctuation can be found incision should be made. Even if no pus is discovered relief may follow from the local blood-letting.

HYPERTROPHY OF THE TONSILLAR TISSUE

There is in childhood a special predisposition to hyperplasia of the tonsillar tissue. The overgrowth as oftenest recognized is in the faucial tonsils, but equally as frequently the pharyngeal tonsil is involved (adenoid growths), and the tissue at the base of the tongue (lingual tonsil) may likewise be affected, although the last is not common in children. Generally the process involves more than one region, although not by any means equally. Probably the pharyngeal tonsil is oftenest attacked to an extent productive of symptoms. The hyperplasia may affect both the lymphatic structure of the tonsil and the connective tissue septa, sometimes the involvement of one predominating, sometimes the other. Often combined with this tonsillar hypertrophy is the enlargement of the follicles visible on the posterior wall of the pharynx on ordinary inspection of the throat.

HYPERTROPHY OF THE FAUCIAL TONSILS

Etiology.—The disease is of very great frequency in early life. Lennox Browne¹ estimates that it constitutes 37 per cent. of all diseases of the fauces and pharynx. Although sometimes beginning in infancy, the hypertrophy is believed not to reach any decided degree until this period is passed. In my own experience, however, enlargement of the tonsils in the 1st year is by no means infrequent. After this period it is extremely common. There is often noted a marked family predisposition, several of the children of the family, and often the parents as well, showing the same tendency to enlargement. The general health usually appears to exert no influence, the subjects of the disease being healthy in other parts of the body. (See Lymphatism, p. 632.) The repeated occurrence of catarrhal pharyngitis is the chief exciting cause. Tuberculosis has no etiological connection.

Pathological Anatomy.—The condition of the tonsil varies with the case. When not associated with a temporary catarrhal inflammation, as shown by the redness of the mucous membrane, the tonsil is large and pale. In some instances the overgrowth is chiefly in the lymphoid tissue, and in these the tonsil is soft and the lymphoid element projects in the form of nodules. If the fibrous overgrowth is in excess the tonsil is hard and firm, broad bands of connective tissue crossing it in different directions. The crypts of the tonsil frequently exhibit yellowish masses, yet without showing the signs of inflammation characteristic of acute lacunar tonsillitis. The hypertrophy is nearly always bilateral, yet one tonsil is often more affected than the other. Very frequently tonsillar hypertrophy is overlooked because the organ is deeply situated and its actual size is not at first discovered. The overgrowth in these cases is

¹ The Throat and Nose and Their Diseases, 1899, 348.

chiefly in depth and width. At the times when an acute inflammation is superimposed, the organ increases much in size and becomes redder.

Symptoms.—Examination shows the presence of enlargement of the tonsils as described. It not infrequently happens, as stated, that the overgrowth has been chiefly in width and depth, and the tonsil is consequently "submerged," being hidden by the anterior pillar and other folds of the soft palate. In such cases palpation with the finger, or causing the patient to gag reveals the degree of enlargement present. In other instances the tonsils may be so large that they almost touch each other. The general symptoms are not well marked, the majority of those formerly attributed to this condition depending in reality upon the adenoid overgrowth which so frequently accompanies the hypertrophy of the faucial tonsils. Moderate enlargement of the latter, if occurring alone, produces practically no symptoms. When, however, the hypertrophy is great there is often a thick tone to the voice as though there were food present in the mouth, and the swallowing of solid food and even the respiration may be mechanically interfered with. As a rule deglutition is not painful. There is a great tendency to repeated, acute tonsillar inflammation, and at this time the symptoms are exaggerated. The lymphatic glands in the neck may become chronically enlarged. Mouth breathing and snoring occur and deafness may result; but, again, these conditions oftener depend chiefly on the accompanying adenoid hypertrophy. The enlargement of the tonsil may frequently be felt externally. In other cases, without symptoms other than the chronic tonsillar enlargement observed on inspection, there is a tendency to repeated attacks of fever without discoverable cause, general impairment of health, and other uncharacteristic symptoms. That these are due, in some instances at least, to a mild toxic condition produced by absorption from a clinically inflamed tonsil, seems proven by the disappearance of symptoms after tonsillectomy.

Course and Prognosis.—The softer lymphoid tonsils vary much in size from time to time, being larger during any acute attack of tonsillitis, and afterward sometimes larger, sometimes decidedly smaller than before this. There is a natural disposition for them to undergo progressive diminution in size as puberty is attained; but unfortunately before this period is reached there exists a constant tendency to an increasing overgrowth of the connective-tissue element, and tonsils of this nature never exhibit any considerable lessening in dimensions. There is always a certain danger of infection taking place through these diseased bacilli tonsils, tubercle bacilli and the germs of the acute fevers entering the system in this way.

Treatment.—With the exception of operative procedure this is on the whole unsatisfactory. Astringent applications may do good in the cases where the lymphoid hyperplasia largely predominates. The administration of cod-liver oil, or of iodide of iron best in the form of a syrup, is sometimes of advantage. Change of residence to a dry climate is often of service. Little or no benefit, however, is to be expected in the cases in which much connective-tissue hyperplasia has taken place. As regards operation, in cases where the hypertrophy is moderate and is producing no symptoms it is well to defer this in the hope of retrograde changes taking place as age advances. Yet, as stated, the presence of hypertrophy of the tonsils always occasions an element of danger. Infection of various sorts, as of tuberculosis, diphtheria, and scarlet fever, is much more prone to take place by this route than in the case of healthy

tonsils. Whenever, then, there is frequent recurrence of lacunar tonsillitis; where slight deafness readily develops; where there is increasing glandular swelling in the neck; where the tonsils are very large; or when, from the constitutional symptoms, there is reason to believe that absorption is taking place, the tonsils should be removed. On the other hand, there has undoubtedly existed a disposition to a too prompt operative removal of the tonsils, often merely on the ground that hypertrophy existed. It is by no means understood just what useful purpose the presence of the organs fulfils, and it is a safe dictum that no operation in any region of the body should be urged, unless there are distinct indications for it. In my own opinion the presence of hypertrophy of the tonsils without other symptoms does not constitute such an indication; and, further, the existence of an etiological relationship of hypertrophy with any symptoms found should appear at least extremely probable. The mere discovery of a few small lymphatic glands in the neck, for instance, is not an indication, inasmuch as this condition is extremely common and may be entirely without significance. There is, it is true, little danger in the operation, although severe and even fatal hemorrhage has been known to occur, and repeatedly septic inflammation of various degrees of severity attacking different parts of the body has taken place. One should also not expect from operation benefit of symptoms which were in reality dependent upon adenoid growths; and the removal of the tonsils does not prevent later attacks of pharyngitis.

For the operative methods to be recommended, reference is made to works on Surgery or on Diseases of the Throat. My own preference is for as thorough a removal as possible, since it not infrequently happens that hypertrophy continues to increase in the portion of the tonsil remaining after a partial removal, or that the deeper-lying diseased lacunæ had not been reached, and that repeated attacks of inflammation continue to take place in these. This removal is accomplished by enucleation. Mention should be made here of the dirty-grey pseudomembrane not infrequently seen upon tonsillotomy-wounds within 24 hours after operation. This is a matter of no significance, although it may readily cause alarm from the resemblance borne to a diphtheritic deposit.

HYPERTROPHY OF THE PHARYNGEAL TONSIL

(Adenoid Vegetations)

Etiology.—This is an extremely common condition in early life, to which attention under the title of “Adenoid Vegetations” was first directed by Meyer.¹ It is found, according to different statistics, in from 1 to 9 per cent. of children apparently healthy in other respects (Ballenger).² Although it is especially in childhood that its symptoms become marked, they are quite frequently observed in infancy also, and the condition may be even congenital. Toward the end of later childhood the incidence diminishes greatly. Children showing evidences of lymphatism are especially prone to adenoid growths. Inheritance is an undoubted factor and a family predisposition is often marked, perhaps all the children of the family presenting well-developed instances of the disease. Dwelling in damp climates; the occurrence of such infectious diseases as measles, scarlet fever, and diphtheria, which are productive of nasopharyngeal inflammation; and repeated attacks of catarrhal dis-

¹Transac. Med-Chirurg. Soc., 1870, LIII, 191. Archiv. f. Ohrenheilk., 1873, I, 241; II, 241.

²Diseases of the Nose, Throat and Ear, 4th Edit. 333.

orders of the nose and throat are also factors, although this last oftener results from the hypertrophy than produces it. Tuberculosis and syphilis have no causative influence; but the possibility of tuberculous changes taking place in the growths and of the entrance of tubercle bacilli into the system is not to be forgotten. (See Tuberculosis, p. 543.)

Pathological Anatomy.—The soft “adenoid” structure resembling that of the faucial tonsils, which is normally widespread in the nasopharynx especially on the posterior wall and the roof, undergoes hypertrophy and forms masses of varying size (Fig. 236) reaching even that of a walnut, and attached to the underlying tissue by a broad base. These may more or less completely fill the vault of the pharynx and cut off the

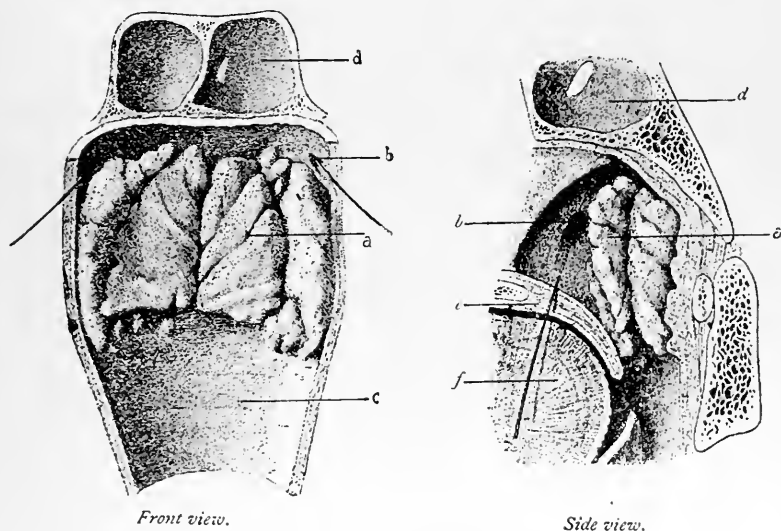


FIG. 236.—ADENOIDS IN SITU.

a, The vegetations; b, Eustachian orifice; c, the pharynx; d, sphenoidal sinus; e, velum of the palate; f, base of the tongue and epiglottis. (Lennox Browne, *The Throat and Nose and Their Diseases*, 1899, 422; after Castex and Lacour.)

passage of air through the choanæ. In young subjects they consist of soft, spongy, lymphoid tissue well supplied with blood vessels, but after a time this grows denser, fibrous and less vascular, resembling more the connective-tissue hypertrophy of chronically enlarged faucial tonsils.

Symptoms.—The manifold symptoms may be divided into those chiefly *local* and direct and those more *general* and remote in nature. Of the former, *mouth breathing* is one of the most characteristic (Fig. 237). The degree and persistence of this varies with that of the obstruction. In the milder cases it may have entirely escaped the observation of the attendants. In some children it is observed only during sleep, especially if on the back, at which time snoring is liable to occur, and this is true even in infancy. In others the obstruction is sufficient to make the sleep very restless and disturbed, the child trying ineffectually to find some position in which respiration may be easier. In cases of decided adenoid hypertrophy the mouth hangs open during the day as well; the lips are dry; the expression dull or stupid; the color of the face sometimes pale. Alteration in the *shape of the nose* is common. The nostrils are small and narrow, and the lack of use of these for the normal purpose of breath-

ing gives the nose a "pinched" appearance (Fig. 238). Sometimes, however, the upper portion of it seems unduly broad. *Nasopharyngeal catarrh* is an attendant symptom either more or less constantly present or occurring in repeated attacks from very slight exposure. The narrowing of the choanæ and the interference with the passing of the nasal secretion backward is an active factor in producing the chronic catarrhal condition. As a result of this the obstruction to respiration is increased; the voice is altered, developing a nasal, muffled character; the breath offensive; and the taste and smell impaired. A harrassing *cough* is often present, especially at night, the result of the irritation of the larynx by the inspired air, which has not been properly warmed and moistened by passing through the nose in the normal manner. In other children the cough may depend upon bronchitis, to which the affected children are greatly predisposed. *Deafness* is a very common symptom, at first temporary and developing with the catarrh; later more or less permanent unless treatment of the adenoids is promptly undertaken. It is the result of the blocking of the entrance of the Eustachian tubes and of the catarrhal processes in them or in the middle ear. The symptoms described depend partly on the permanent obstruction by the adenoid growths and partly on the associated catarrhal condition. Consequently they vary from time to time, being on the whole better in summer and worse in the winter season.



FIG. 237.—ADENOID FACE.

(Thomson, *Clinical Examination of Sick Children*, 1908, 13, Fig. 9.)

Permanent *bony deformities* of the structure of the skull may be the result of the pressure of the adenoid tumors or the efforts at respiration. There is seen, namely, in some cases a high and narrow arching of the palate, and irregularity in the position of the teeth of the upper jaw. *Exophthalmos* is sometimes observed. One of the most marked deformities of this sort is the alteration of the thorax, oftenest in the form of the keel-shaped chest with lateral depression of the lower portion of the thorax. This occurs in patients who have developed adenitis early, and is the result of the unusual efforts at respiration required to obtain a sufficient amount of air in the lungs. It is best seen in patients also the subject of rickets. *Scoliosis* may also result. *Enlargement of the cervical lymphatic glands* is nearly always present at some time in well-marked cases.

But the list of symptoms is by no means complete with those mentioned, and a number of more or less indirect or general results are evident. There is often a decided *retardation of mental development*, sometimes the result to a certain extent of the impairment of hearing. The stupid expression, dependent in part upon this mental state and in part upon the direct mechanical effect of the adenoids in keeping the mouth hanging open and in producing deafness, may, however, give only a mistaken idea that the patient is imbecile. The *growth of the body* is also often interfered with, anemia is common, and the general health suffers. Among other conditions sometimes associated with adenoid hypertrophy and relieved by its removal, are stammering and stuttering, laryngospasm, spasmodic croup, bronchial asthma, hoarseness, headache, night terrors, enuresis, chorea, grinding of the teeth and convulsive attacks.

Course and Prognosis.—Adenoid growths are disposed to increase gradually in size and the symptoms consequently to grow worse, reaching their height about the beginning of later childhood and continuing until puberty unless relieved by treatment. At puberty there is a tendency for the growths to become smaller and for symptoms to disappear, unless the tissue has become of a fibrous nature. So, too, the condition is always better in summer time and in a dryer climate, due to the diminution of the catarrhal involvement of the mucous membrane and the shrinking of



FIG. 238 —DEFORMITY FROM ADENOID GROWTHS.

Child of 7 years and 9 months. Shows the narrow nasal bridge and small nostrils, as well as a marked degree of funnel-chest from persistent respiratory tugging.

the growths; worse in winter and during the occurrence of an acute nasopharyngeal catarrh. Owing to this disposition to grow worse, there is a likelihood of serious deformities and affection of the health developing. This is particularly true when symptoms have appeared during infancy. Infection of various sorts, especially by tuberculosis and diphtheria, is liable to occur by way of the adenoids, and the presence of these vegetations makes many diseases affecting the throat run a more serious course. The results from operative treatment are sometimes remarkably prompt and complete; oftener somewhat slower in manifesting themselves; sometimes disappointing if the adenoids have been allowed to remain too long and if bony deformities or nervous symptoms have developed.

Diagnosis.—In well-marked cases this presents little difficulty. The presence of the characteristic obstructive symptoms described is most suggestive, especially the mouth-breathing and snoring, the expression of the face, occurrence of deafness, and the obstinate or frequently recurring nasal catarrh. The existence of hypertrophied faucial tonsils indicates that adenoids are very probably present also. The diagnosis can be confirmed by digital examination of the vault of the pharynx. This reveals the irregular, soft, nodular masses, or sometimes a tough, firmer growth. Slight bleeding nearly always follows the examination. In older children the masses may be seen by rhinoscopic examination. Even in patients suffering from vague symptoms, such as headaches, anemia, debility, various nervous manifestations, mental backwardness, and so on, one should make sure whether or not it is the presence of adenoids which perhaps accounts for the condition.

Treatment.—Owing to the tendency to increase in size and the many dangers which attend the presence of adenoids, the best treatment is undoubtedly their early and thorough removal. Delay may be made, however, where the growths are small and but little obstruction occurs, and where the symptoms can be relieved by change of climate, especially in winter, or by successful measures to prevent catarrhal attacks. Such patients should, however, be under constant medical supervision. In the meantime efforts should be made to maintain and improve the general health by tonic remedies, especially cod-liver oil. Positive indications for prompt operative interference are continuous mouth-breathing, affection of speech, otitis, deafness, beginning bony deformities, retarded mental or bodily development, and persistent or constantly recurring nasal catarrh and bronchitis. Asthma, cervical adenitis, enuresis, night terrors, headache, and other nervous symptoms not relieved by other treatment often make adenotomy advisable.

Although operative removal is such an effectual treatment in this disease, it should not be practised merely as a routine measure, but determined for each individual case. To operate needlessly is as much a fault as is the failure to operate when indicated. The age for operation is the time when serious symptoms are threatening, even in infancy. When, however, it can be deferred without danger it is better to wait until infancy is past. So, too, spring or summer is the season to be elected for operation when this can be managed without detriment, since catarrhal nasal disorders are less likely to complicate the condition at this time. Operation should not be performed during an attack of rhinitis. Thorough removal is a bloody and sometimes a rather tedious operation, to be performed under ether. It is, however, in my experience, much to be preferred to a rapid partial removal, since recurrence of the growth is very likely to follow the latter procedure. The operation should be done only by one giving especial attention to this branch of surgery, and details may well be omitted here. Excessive and even fatal hemorrhage has occurred either during the operation or afterward, and nephritis has sometimes developed. I have seen several instances of this. Sudden death under the anesthetic has also been seen, due doubtless to the accompanying lymphatism frequently present. After operation the denuded surface affords a ready portal of entry for diphtheritic, scarlatinal or septic infection. On the whole, however, these accidents are uncommon and the danger of operation is very slight.

CHAPTER IV

DISEASES OF THE ESOPHAGUS

MALFORMATIONS OF THE ESOPHAGUS

These are occasionally seen in early life, and most of them are of congenital origin. In conjunction with Dr. R. S. Lavenson, I have reviewed the subject to some extent in a previous publication¹ where a bibliography may be found. Some of these malformations are incompatible with life and the infant dies a few days after birth; others may be continued indefinitely. A statistical review of the subject has been published by Cautley.² Among the malformations may be mentioned:

1. **Branchial Fistulæ and Cysts** are the result of a failure of complete closure of the branchial cleft, which opens in fetal life through the neck into the upper part of the esophagus or the lower portion of the pharynx. It consists of a small, external fistula usually unilateral, and oftenest just above the sternoclavicular articulation; sometimes high in the neck at the inner edge of the sternocleidomastoid muscle. The fistula ends blindly or may communicate with the alimentary tract. Treatment is usually unsatisfactory and had better not be attempted except in those cases where there is a continual mucous discharge, or where the external opening of the fistula has become clogged and a disfiguring cyst-like mass results. Cysts of a similar appearance, due to other causes, occur in this locality. To all such growths the title *Hygroma* is often applied (Fig. 239).



FIG. 239.—HYGROMA CYSTICUM.

Patient aged 7 months, in the Children's Hospital of Philadelphia. Tumor was noticed at birth, grew steadily larger, and finally interfered with respiration and deglutition.

2. **Diverticula of the esophagus** are occasionally seen. It is doubtful whether they are ever really congenital. They occur most frequently as the result of traction exerted by adhesions of the esophagus to the trachea or to a bronchial gland. Their situation is oftenest at the level of the bifurcation of the trachea. The food taken, especially if solid, experiences difficulty in passing into the stomach, and often is regurgitated after a shorter or longer interval without nausea and without evidences of action of the gastric secretion. In some cases swelling upon one side of the neck is present when the diverticulum is distended by food. The sound when passed may catch in a pocket somewhere in the course of the esophagus, or may at other times pass into the stomach. The employment of the x-ray after the administration of bismuth serves to confirm the diagnosis of diverticulum.

3. **Congenital absence of the esophagus** is very rare. There were found but 7 reported cases.

4. **Congenital stenosis** is uncommon. It is due either to a fold of mucous membrane or to narrowing of the entire wall of the tube. It is

¹ Arch. of Pediat., 1909, XXVI, 161.

² Brit. Jour. Child. Dis., 1917, XIV, 1.

attended by the symptoms of stenosis seen in the acquired form. (See p. 694.)

5. **Dilatation of the esophagus** as a congenital lesion is limited to the portion just above the diaphragm. A consequent acquired secondary diffuse dilatation of the entire length of the esophagus may result. Acquired dilatation is liable also to be a sequence to stenosis of any nature, but is oftenest seen in corrosive esophagitis with stricture. (See p. 694.)

6. A partial or complete **doubling of the esophagus** has been observed in 2 or 3 instances.

7. Sometimes a **tracheo-esophageal fistula** exists without other lesions. This is very rare. (See below).

8. Finally there may be a congenital **obliteration of the lumen** of the esophagus in a portion of its extent. Unattended by fistula this is very uncommon. We found but 17 published cases,¹ but a number of others have since been reported. The most common congenital malformation of the esophagus is a combination of obliteration through more or less of its extent with tracheo-esophageal fistula, connecting the trachea just above the bifurcation with the portion of the esophagus below the closure (Fig. 240). The upper portion of the esophagus is somewhat dilated and ends blindly at the point of obstruction. The chief symptom of obliteration is the complete inability to swallow food; it being promptly regurgitated through the mouth and nose, producing severe suffocative attacks. Attempts to pass a sound encounter the obstruction.

SPASM OF THE ESOPHAGUS (Esophagismus)

Although a disease oftenest seen beyond puberty and in women, cases occasionally occur in children, and I have encountered 1 very typical instance.² A neurotic or psychopathic element is present, and the condition is much influenced by observation, sympathy, and the like. The difficulty in swallowing may be constantly present, or may occur only at intervals under increased nervous excitement. In some cases liquids

can be taken readily, although solids give trouble; while in others there is difficulty with both sorts of nourishment. Sometimes hot liquids can be swallowed better than cold. The diagnosis is to be made especially from organic stricture by the fact that an esophageal bougie passes without difficulty, at least under anesthesia. The prognosis is good so far as life is concerned, but the duration of the disorder is often long. Treatment should be directed especially to the nervous system. Sympathy and over-anxiety should not be shown by the parents; a skillful nurse

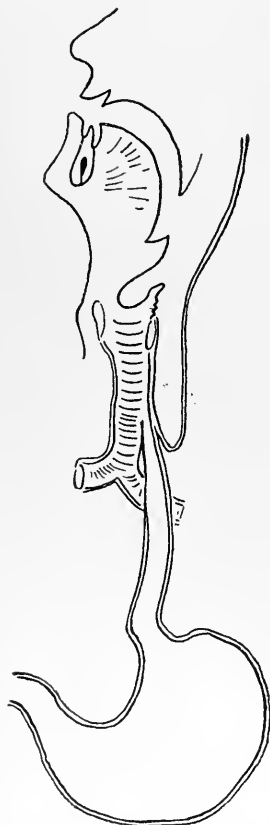


FIG. 240.—DIAGRAM
ILLUSTRATING TRACHEO-
ESOPHAGEAL FISTULA.

(Griffith and Lavenson,
Trans. Amer. Pediat. Soc.,
1908, XX, 86.)

¹ *Loc. cit.*

² *New York Med. Journ.*, 1914, XCIX, 113.

is of great aid; and the patient does better if removed from home. General tonic and hygienic measures, change of air, and the like are serviceable. It may be well to pass a bougie daily.

CATARRHAL AND FOLLICULAR ESOPHAGITIS

Acute catarrhal esophagitis may occur under a variety of conditions, and may be seen even in the new born (Billard).¹ It may occur in the course of the acute infectious diseases or pneumonia; attend catarrhal inflammation of other parts of the digestive tract; or follow lacerations produced by the swallowing of foreign bodies or the injury done by the ingestion of hot liquids. The mucous membrane is injected and swollen and the subcutaneous connective tissue edematous. Superficial erosions may occur in severe cases. The lesions, as far as known, generally last but a few days, and the prognosis is favorable. Symptoms are absent or are uncharacteristic and consist of mild pain on swallowing.

Chronic catarrhal esophagitis is of unusual occurrence in early life. It may follow an acute catarrhal process, or be the result of venous congestion in chronic pulmonary or cardiac disease.

Follicular esophagitis is probably more uncommon than the catarrhal variety. It has been found in typhoid fever and in chronic gastrointestinal and respiratory diseases. The lesions consist in enlargement of the mucous follicles, sometimes attended by superficial erosion of them.

A **secondary esophagitis** of a different nature from the lesions described may occur in the course of various diseases, but is rare. Thus diphtheritic or other pseudomembranous inflammation has been found in the esophagus; thrush may extend into it and even obstruct its lumen; pustules of variola have been discovered there; and ulceration may result from perforation of a caseous lymphatic gland. As with the other forms of esophagitis mentioned, the recognition is usually impossible during life.

CORROSIVE ESOPHAGITIS

(Stricture of the Esophagus)

Corrosive inflammation is the variety of esophagitis oftenest seen in children. The most frequent **cause** is swallowing of caustic solutions such as strong acids or alkalis, given to the children by mistake, or carelessly allowed to be within their reach. The **lesions** depend upon the strength and amount of the fluid ingested and the degree of penetration of the corroding process, this varying from a superficial necrosis of the epithelium to a destruction of the entire thickness of the mucous membrane. In the severer cases a slough results which finally separates, and, if the patient survives, is at last replaced by cicatricial connective tissue, resulting finally in stenosis as contraction takes place. In the worst cases even perforating ulceration of the esophageal wall occurs.

Symptoms.—The early symptoms are immediate pain and burning from the mouth downward, with vomiting of bloody mucus; painful or impossible deglutition; and great thirst. Total collapse may occur at once, and death take place in a few hours or days; or somnolence and fever may mark the severity of the toxic action. If the danger of early death is passed, there remains for some days the evidence of severe local inflammation with great pain especially on swallowing. The mucous membrane of the mouth is in places denuded of epithelium. Vomiting of pieces of necrosed mucous membrane may occur. Later death may

¹ *Traité des mal. des nouveaux-nés*, 1828, 274.

result from perforation into the peritoneal or the pleural cavity. If this does not occur, improvement gradually follows, although erosion of the blood-vessels or perforation of an ulcer may later unexpectedly take place. After an interval usually of some weeks or months the symptoms of **stricture of the esophagus** begin, if the corrosive action has been more than merely superficial. There is then an increasing difficulty experienced in swallowing solid food, which often is regurgitated after the attempt is made. Finally, in severe cases, even liquid food is ejected, and examination with the sound shows a more or less complete stricture of the tube. The position of the stricture and its character vary. It may be annular or cylindrical in form. In the majority of cases in children it is situated in the upper third of the esophagus. Torday¹ found it in the upper third in 54 per cent. of his cases, and in the middle third in 19 per cent.

It should be borne in mind in this connection that, although the great majority of cases of stricture of the esophagus are dependent upon corrosive esophagitis, there are exceptions of various sorts. Congenital atresia or stricture of the esophagus has already been referred to (pp. 691, 692). There may further be mentioned stricture from the lacerations produced by foreign bodies, and a spasmodic stenosis occurring in hysteria or rabies; while diphtheria, syphilis, and variola have produced the disease by ulceration. Narrowing by compression from without may occur in cases of retroesophageal abscess, inflammatory enlargement of the thyroid gland and caseous tracheobronchial glands.

After the development of cicatricial stricture a **dilatation of the esophagus**, either cylindrical or sacculated, generally forms above it and food may lie for some time in this before being regurgitated. If the stricture is decided, rapid loss of weight and of general health will occur. There is, however, frequently a certain degree of variability in the completeness of the obstruction, and children may be able to swallow fairly well at certain times and not at all at others. This probably depends upon a varying increase and decrease in the swelling of the mucous membrane.

Prognosis.—The prognosis of corrosive esophagitis is always serious and uncertain. A large number perish from the primary lesion, and of those who survive a considerable part die of stricture. Torday reporting on 208 cases of corrosive esophagitis in children produced by the ingestion of lye, found that 172 (82.69 per cent.) later developed the evidences of stricture; the percentage being the same as occurred in the experience of Keller² (82 per cent.) many years earlier. Cases where the lesions of the mouth and pharynx soon heal probably also have an involvement of the esophagus which is likewise mild, and the prognosis is consequently better.

Treatment.—The first indications for treatment, if the child is seen early enough, are the exhibition of antidotes to the corrosive poison and the washing out of the stomach. After this the administration of ice and of demulcent solutions internally is indicated, such as oil, flaxseed or gum arabic solution, and the like, with morphine hypodermically to relieve the suffering. Any food given must be liquid and perhaps best administered by rectum if the child will retain it. In cases in which stricture has developed, the treatment is purely surgical and consists in the systematic and very careful use of bougies of a progressively increasing size,

¹ Jahrb. f. Kinderheilk., 1901, LIII, 272.

² Oesterreich. Zeitsch. f. Heilkund., 1862, VIII, 856.

beginning with small instruments when necessary. This procedure should not be commenced until at least 3 or 4 weeks have elapsed, and all acute symptoms have subsided. If no bougie can be passed, gastrostomy becomes necessary, followed by an attempt to dilate the stricture from the cardia. The use of bougies must be continued for months in order to prevent recurrence of the stricture.

FOREIGN BODIES IN THE ESOPHAGUS

With the disposition of children to put all sorts of objects in the mouth, the lodging of foreign bodies in the esophagus is of not infrequent occurrence. Careful examination with the esophageal sound will reveal the presence of the body and its position, and the use of the Röntgen ray is of great aid in confirming the diagnosis. The place of lodgment is oftenest either at the upper opening, or lower where the left bronchus crosses the tube. The removal of such bodies has become a strictly surgical procedure, the object being pushed into the stomach, or withdrawn through the mouth. Skill is required to avoid doing injury. Sometimes esophagotomy is required. Should the object pass into the stomach amylaceous food, such as potato and cereals, should be given freely in order to render the feces consistent and thus coat the foreign body and protect the bowel from injury.

RETROESOPHAGEAL ABSCESS

(Periesophageal Abscess)

This rare condition is sometimes seen in infancy and childhood. But 1 instance has come to my observation, and a study of the literature in 1898¹ found but 12 others reported in detail up to that time.

Etiology.—The causes are similar to those producing retropharyngeal abscess, except that spinal caries appears here to be the most frequent etiological factor. Among other causes which may induce periesophageal inflammation are pleuritis and pericarditis; ulceration resulting from a foreign body in the esophagus, or from a tracheotomy tube or intubation tube; diphtheria of the pharynx; and suppurating lymphatic glands. The abscess forms behind and around the esophagus and often displaces it readily to one side, while it exercises compression upon the more firmly seated trachea or on other parts.

Symptoms.—These are very uncharacteristic and misleading. Dyspnea is nearly always present and is the most prominent and urgent symptom. Cough, too, is generally observed; sometimes only slight, sometimes spasmodic or brassy and suggesting stenosis. Dysphagia might be expected, but was absent in all the reported cases in my series. This is doubtless due to the fact that the esophagus yields easily to the pressure and changes its position. Swelling in the neck may result if the abscess is situated behind the upper part of the esophagus. Affection of the voice is uncommon and, in any event, does not present the peculiar characteristic nasal alteration observed in retropharyngeal abscess.

Prognosis.—The prognosis is nearly always unfavorable. Death may result from the pressure of the abscess upon the pneumogastric nerve, or on the trachea with consequent asphyxia; or rupture into the trachea, bronchi or lung may occur and a purulent bronchitis or broncho-

¹ Univ. Med. Mag., 1898, Jan.

pneumonia follow. Even should the abscess discharge itself into the esophagus, asphyxia from the pus being regurgitated and entering the larynx in large quantities may take place. If this passing into the larynx is escaped, recovery may follow. Unfortunately the spondylitis which is the commonest cause of abscess remains, suppuration continues, and the danger of pus entering the larynx exists for every future occasion of the discharge of it in large amount.

Diagnosis.—This is always difficult and generally cannot be more than conjectural, since other pathological conditions may produce very similar symptoms. If caries of the vertebra is known to exist, retroesophageal abscess may be suspected if the dyspnea and other symptoms as described develop. If the abscess has a very high situation it may perhaps be reached by the finger thrust deeply downward through the pharynx, or a lateral swelling in the neck may be visible. If there is a history of the swallowing of a foreign body, employment of the Röntgen ray may reveal the presence of an abscess.

Treatment.—Therapeutic measures can seldom be employed. If the abscess is situated high enough to be discoverable by the finger it may be opened. Otherwise nothing directly in the way of treatment can be done. Tracheotomy may be performed if there is urgent dyspnea; not with the hope of relief if the condition is due to retroesophageal abscess, but because, with the uncertain diagnosis, some other disorder may be the cause of the respiratory stenosis.

CHAPTER V

DISEASES OF THE STOMACH AND INTESTINES

In the absence of a fully complete knowledge of the physiology of digestion and of the pathological lesions found in the digestive tract, any classification of the diseases of the stomach and intestines can be only provisional. That based either upon the lesions alone or upon etiology alone is no more practically useful than is that scientific which rests solely upon symptoms; since the same lesions may be productive of different symptoms, and, on the other hand, identical symptoms may be the result of quite diverse pathological causes. There are diseases, too, which appear to depend entirely, or in great part, upon functional disturbances rather than upon pathological lesions, and it is often difficult or impossible to determine from clinical manifestations how far the disordered condition is due to one or the other factor. The rôle of microorganisms and their toxins is also important in the production of gastrointestinal disease; the bacteria being either those foreign to the alimentary canal, or those which are natural inhabitants of it under ordinary circumstances, and are then without harmful influence. This rôle is, however, uncertain; sometimes the affection being probably chiefly a disturbance of function through the poisonous toxins; sometimes a distinct pathological infection produced by the germs; although just which action predominates cannot well be determined. Indeed of recent years the influence of germs in producing gastrointestinal disorders has been largely called in question in many quarters, and many of the disturbances of this sort are assigned to metabolic processes depending often upon the nature of the food given.

Again it is evident that the diseases of the stomach and of the intestine respectively cannot be always sharply separated from each other, since

so often the symptoms of both may appear simultaneously or consecutively, or sometimes those of the one region predominating and sometimes of the other. With these facts in view the difficulties attending any classification are manifest.

THE FINKELSTEIN CLASSIFICATION

The theories of Finkelstein regarding nutritional diseases associated with disordered digestion or metabolism; an elaboration and modification of the views of Czerny and Keller¹ have attracted so much attention that a review of them is necessary. The classification is given at length in the article by Finkelstein and Meyer in Feer's work on Pediatrics² and previously in many journal-publications. (See also reviews by J. Hess,³ Meara,⁴ and others; and especially by Snow.⁵) Instead of classifying the disorders according to the portions of the digestive tract exhibiting symptoms, or viewing them as dependent upon bacterial action, he regards them rather as disturbances of metabolism, the result of the toxic action of substances derived from the different normal elements of the food, given in a combination unsuited to the child. The symptoms, then, are evidences of intoxication rather than of infection. The element of decomposition of the food by the action of bacteria, as in impure milk, he considers as of minor importance. The healthy breast-fed baby has a normal tolerance for food; but the artificially-fed infant has to deal with food-ingredients not natural to it, and a degree of intolerance for some of these is readily established. This is especially true if the child is weakly, or the digestive functions disordered. There is a loss of the natural balance between the food required and the ability of the child to assimilate this. The casein of the milk is, according to his view, the ingredient most readily borne. The whey, on the other hand, is a source of much trouble. The sugar and the fat readily produce intolerance, but chiefly when given in combination with or contained in the whey. Abnormal fermentation of the food in the digestive tract occurs, for instance, during hot weather from a diminution of the power of digestion rather than from unusual contamination by bacteria.

The development of intolerance may be shown by various symptoms, their nature depending upon the severity of the process. These symptoms are not only those distinctly digestive, such as vomiting, diarrhea, and the like, but others of deranged metabolism producing nervous manifestations, albuminuria, fever, etc.

Finkelstein divides the disturbances into: (1) Disturbance of balance; (2) Dyspepsia; (3) Decomposition, and (4) Intoxication.

1. Disturbance of Balance.—This is the mildest modification of the food-intolerance. The most common injurious element of the food is the fat. This is not necessarily because the fat is in an unduly large amount, but because the increased alkaline secretion of the intestines combine with the fatty acids producing soap-stools. The chief symptoms are unsatisfactory gain in weight in spite of the administration of food the caloric value of which is sufficient. There is to some extent a "paradoxical reaction"; *i.e.* a greater loss of weight if the food is increased.

¹ Des Kindes Ernährung, Ernährungstörungen und Ernährungstherapie, 1906, II.

² Feer, Lehrbuch der Kinderheilkunde, 1914, 223.

³ Amer. Jour. Dis. Child., 1911, 11, 422.

⁴ Arch. of Ped., 1910, XXVII, 579.

⁵ Arch. of Ped., 1909, XXVI, 801. Amer. Journ. Dis. Child., 1914, VIII, 163.

There are also occasional vomiting and tympanites and often firm soap-stools, but no special signs of illness. The most effective treatment is a diminution of the fat and an increase of the carbohydrate element of the diet.

2. Dyspepsia.—This is also a mild form of the disturbance. Its commonest cause is an inability to assimilate carbohydrates, with a consequent fermentation of these in the intestine, and resulting increase of peristalsis, with diarrhea. This fermentation of the carbohydrates prevents the proper absorption of the fat, which then appears in the stools. Increase in the amount of food produces loss of weight (paradoxical reaction). The symptoms consist in the same failure to gain weight seen in disturbance of balance, together with the occurrence of diarrhea. There is loss of appetite, vomiting, tympanites and colic. The stools are thin, green, frothy, and contain mucus and white lumps composed of fat and bacteria. Treatment is best carried out by giving human milk. In the absence of this the carbohydrates should be reduced after a brief period of fasting. Often the employment of some other sugar than that of milk is successful; cane sugar and, still better, dextrine-maltose preparations being less liable to ferment. The fat also should be reduced. Buttermilk is often of service. He regards "casein-milk" as one of the best of foods for the condition. (See p. 148.) Too long a continuance of under-feeding must be avoided.

3. Decomposition.—By this term is indicated a loss of the constituents of the body; a decomposition. It is one of the severe forms of nutritional disorder, the equivalent of the condition ordinarily described as infantile atrophy, or marasmus. It depends upon a very decided loss of digestive power. There is a great intolerance for fat and carbohydrate. The symptoms consist in gradual and progressive loss of weight, and the infant has the ordinary appearances of marasmus elsewhere described (page 610). The temperature is subnormal, the abdomen distended, the color pale, the child is at first excited but later torpid, the movements of the body are slow, and the appetite is often large. The stools are not well digested, and may be either solid or diarrheal, and often contain an excess of fat. The pulse is slow; the respiration often irregular. There is a very decided paradoxical reaction, more marked than in the preceding forms; any increase in the amount of food given being at once followed by decrease of weight. The system is very susceptible to infection and other morbid influences, and fever is produced readily from such causes or from an increase in the food. Edema and cyanosis develop readily. The prognosis is on the whole unfavorable, although under suitable management recovery may occur, if the condition is not too far advanced. Sudden death is not uncommon, while other children die with the symptoms of the alimentary intoxication to be described. Treatment consists in stopping the abnormal changes going on in the food and giving sufficient suitable nourishment. This can best be accomplished by feeding with human milk; often preferably from a bottle and diluted. If human milk cannot be obtained, the most suitable foods are skimmed milk or buttermilk fortified by a starchy addition; and especially casein-milk. The last is claimed to be of particular value on the ground that by the removal of the milk-sugar which is contained in the whey the tendency to fermentation is lessened.

4. Alimentary Intoxication.—This is a threatening condition of food-intolerance developing as a later stage after that of dyspepsia or of decomposition. The disorder is probably an acidosis. The cause is

the administration of food much above the tolerance of the child, and the consequent severe disturbance of metabolic processes. Food rich in whey or carbohydrate is especially prone to produce the condition; but excess of fat is toxic also. The first symptom is fever; proven to be alimentary in that it ceases promptly when food is withdrawn, unless some complicating infection is present. The degree of fever varies with the severity of the case. It may be the only early symptom and be moderate or high, or in cases following decomposition there may be no elevation of temperature. With the fever may be diarrhea and loss of weight. In well-marked cases of the fully developed condition nervous and mental disturbances are among the most prominent symptoms. There may be only lassitude and sleeplessness, or there may be deep coma with sunken eyes, or convulsions or other meningeal symptoms. Vomiting is common, often severe and sometimes violent; and when there is diarrhea the stools are alkaline, but variable in other characteristics. Loss of weight is very rapid on account of the removal of liquid from the system, and the skin becomes shrivelled, the fontanelle sunken, and the muscles of the trunk and limbs often painfully contracted. The same loss of fluid probably accounts for the ready development of collapse. The urine contains albumin and often hyaline and granular casts, and there may be alimentary glycosuria. There is always leucocytosis even up to 30,000. The prognosis depends upon the previous condition and the severity and duration of the symptoms; but on the whole is not unfavorable in cases developing acutely and treated promptly, and not antedated by severe decomposition. The treatment consists in the immediate cessation of all feeding and the administration in some way of large amounts of water, adding saccharin if sweetening is necessary. Starvation cannot be long-continued in marantic cases. In returning to food, human milk should be obtained if possible; if not, fat and sugar should be at first avoided. Casein-milk and buttermilk are recommended.

The advantage of Finkelstein's classification is that it tends to a unification of many gastrointestinal disorders, the division of which into separate entities has long been a problem without satisfactory solution. Among its disadvantages is its assumption as finally proven of something which is by no means universally admitted. Those who have long followed the increasing purification of the milk-supply, as connected with the lessening morbidity and mortality of infants during hot weather, find it difficult to admit that bacteria play as small a part in the production of summer-diarrhea as Finkelstein's claims assign to them. There are, also, sufficient grounds based on experimental work to render it probable that in many cases a change in the character of the food does good by militating against the growth of certain harmful species of bacteria in the intestinal canal, whether or not these possess any distinct infectious power. Then, too, the study of metabolism and its relation to the character of the food given is a subject undergoing constant modification as new facts are discovered, and the results obtained are far from being uniform. It is in accord with the statement of the opening part of this chapter to say, that in the present state of our knowledge it is as impossible to classify the disorders of the stomach and intestine on a purely metabolic theory as it is upon a purely bacteriological one. On this account I have attempted to adopt provisionally, and as a matter of convenience, a classification based chiefly upon the clinical manifestations as connected with the different regions of the gastrointestinal tract; modified by what we may believe we certainly know of the respective action of the food-ingredients and of the bacteria.

VOMITING

Vomiting, although in no sense a disease itself but a symptom of many other affections, is so common in infancy and childhood that a review of some of the causes and varieties is in place. (See also p. 206.)

1. One of the most troublesome forms seen in infancy is due to *overloading of the stomach*. This may occur simply from overfilling of the organ in a child in other respects healthy. At first it is a mere regurgitation of the excess of food taken; is harmless in itself; and is unattended by evidences of nausea, such as sudden cessation of crying, pallor about the mouth, and the like. If the overloading is persisted in the condition may soon become worse, an acute or chronic dyspeptic condition develops, and vomiting empties the stomach more or less completely.

2. What may be called *nervous vomiting* or *habit-vomiting* is an exceedingly intractable and common form seen in many infants. In such cases the slightest excitement may produce vomiting; such as crying, sudden movement of the infant by the mother or nurse, or sometimes the mere psychic stimulation connected with taking food, laughing, or even smiling. It is on account of the mental influence that vomiting may be almost or quite absent during the sleeping hours, but may occur after every feeding during the daytime. Doubtless a more or less dyspeptic state is present in most of these cases. A very similar condition is often seen in older children, in which vomiting becomes a habit, and anger or other emotion may readily produce it. Such children vomit when any medicine is given which has an unpleasant taste, and sometimes if the administration of even an agreeably tasting medicine is attempted, or if food is urged of which they are not fond. Many of these children seem to have the power of vomiting at will, and take advantage of this to the overthrow of all discipline. In other instances the vomiting is dependent upon a neurotic or hysterical condition. Some cases of this nature vomit especially in the morning, before or after breakfast, the occurrence being connected with the excitement or overwork of school-life. Still another cause of nervous vomiting depends upon a disturbance of the equilibrium of the body, as in swinging, sea-travel, or railroad-journeys.

3. *Organic nervous diseases* are frequently attended by vomiting dependent upon actual pathological changes outside of the stomach. This is especially true of meningitis and of intracranial tumors and abscess. It is often the earliest symptom noticed and is sometimes very persistent, and often violent and "projectile" in character. The other evidences of intracranial disease soon develop.

4. Vomiting is nearly always present in *acute gastric dyspepsia*, due to the ingestion of indigestible food, whether in infants or older children. It may come on immediately after eating, especially in infancy, but usually occurs after some hours, the contents of the stomach then showing evidence of disordered digestion. It is preceded by nausea, faintness, loss of appetite, and sometimes fever, and may continue after the first emptying of the stomach; mucus and finally bile appearing in the vomited matter.

5. *Chronic gastritis* is attended by the frequent vomiting of food which has remained too long in the stomach and has undergone decomposition, or of merely an acid, watery liquid.

6. *Acute infectious diseases* are very commonly ushered in by vomiting. This is especially characteristic of scarlet fever and pneumonia, but is also true of malaria, typhoid fever, poliomyelitis and other infectious

disorders. It probably is caused by the direct action of the poison of the disease.

7. *Toxic vomiting* may be produced in various ways. It is seen for instance in recurrent or cyclic vomiting, being dependent upon some poison in the blood. In a similar way uremia is often productive of vomiting. Poisonous substances introduced into the stomach may cause vomiting by direct irritation of the organ or, as in the case of contaminated milk, by the actual absorption of poisonous material produced by the changes which have taken place in the food. The vomiting which often occurs in the later course of diphtheria, typhoid fever and other infectious diseases is probably toxic or septic in nature.

8. *Vomiting after cough* is seen especially in pertussis, but may occur after severe coughing from any cause.

9. *Obstructive vomiting* is observed in several conditions. One of the most frequent causes is intestinal obstruction, especially intussusception. It is attended by constipation, prostration, and in intussusception by bloody mucous evacuations. Rarely the vomited matter becomes fecal later in the disease. Congenital obstruction of the duodenum or pylorus is a cause of obstinate vomiting in early infancy.

10. *Appendicitis* is commonly productive of vomiting attended by severe abdominal pain early in the attack. Later it may recur from the development of septic poisoning.

11. General *peritonitis* from any source is nearly always attended by vomiting. It is accompanied by the other signs of the disease and is due either to sepsis or to paralysis of peristalsis, which consequently produces practically an intestinal obstruction.

12. *Passive congestion of the stomach*, such as occurs in severe forms of heart-disease, is often attended by vomiting with evidences of chronic indigestion.

13. *Reflex vomiting* may depend upon many causes, as the presence of worms in the intestinal canal, the putting of its fingers by the infant into its mouth and throat, and the like.

In every case of vomiting the important and often difficult matter is to determine the cause. Treatment is then that indicated for this.

RECURRENT VOMITING

(Cyclic Vomiting)

This type of vomiting has symptoms so peculiar and characteristic that it may properly be described as an independent disease. Cases of this nature had been reported earlier, but the first important description of the condition appears to have been by Gruère,¹ and it has only in recent years been carefully studied, especially in this country and in France. The title "recurrent" is, I think, to be preferred to "cyclic," since the latter implies a certain regularity which is not a characteristic of the disease.

Etiology and Pathogenesis.—This has been much discussed, yet is but little understood. The first attack usually develops in early childhood; occasionally in infancy. Sex exercises no important influence. A highly developed nervous organization seems to predispose, but the disease is by no means confined to such children. It is noteworthy, however, that it occurs nearly always in private practice. Family

¹ Précis des travaux, de la soc. méd. de Dijon, 1838-1841. Ref., Northrup, in Grancher and Comby, Traité des malad. de l'enfance, 1904, II, 191.

history is without special influence except in the predisposition of children inheriting a nervously organized constitution. Fatigue; the development of some minor disease; and exposure to cold or to fright or other emotional disturbance, seem sometimes to precipitate attacks. Diet undoubtedly has some influence, but the nature of this is not entirely understood, since although an alteration of it seems effectual in preventing attacks in some cases, the most careful regulation is without result in others. The occurrence of slight premonitory yet distinctly digestive symptoms certainly suggests an etiological relationship.

Various theories have been advanced to explain the pathogenesis of the disease. One maintained especially by French writers is that the affection is a manifestation of the uric acid diathesis, or "arthritis." The production of vomiting by this condition is not, however, as yet proven. Marfan¹ and others in France have associated recurrent vomiting with acetoneuria and designated it "acetoneuric vomiting," on the ground that acetone is so constantly present in the urine. There is no evidence, however, as Marfan admits, that acetoneuria produces the attack, since acetoneuria is seen in so many other affections, and may well in this disease be the result of the original cause or sometimes of the starvation-process. The presence of β -oxybutyric acid in the urine, pointed out by Edsall,² indicates the possibility of the condition being an acidosis. This view has much in its favor, but is still wanting certain proof, and the difference between acidosis and the mere presence of acetone bodies in the urine is to be borne in mind. (See p. 635.) Sedgwick³ and Mellanby⁴ found a urinary excretion of creatin at the time of the attack, and believe this points to abnormal metabolic changes. The former also attributed to adenoids a powerful etiological influence. The occasional occurrence of icterus has led others to believe that the liver is at fault (Richardière).⁵ Comby⁶ thought that the vomiting depended in many instances upon a chronic appendicitis. That the attacks are of hysterical origin has been maintained, but the occurrence of fatal cases with evidence of renal lesions renders this unlikely, and certainly inapplicable to all instances. Snow⁷ believed it dependent in some cases upon an intermittent hyperchlorhydria. My own experience leads me to the opinion that the disease is a toxic neurosis occurring in those especially predisposed to it, and that the outbreak depends upon the gradual heaping up in the system of a poison of a nature as yet undetermined, yet it may be an acid arising possibly in the digestive tract or more probably in disordered metabolic processes, and that inability to assimilate the fat of the diet is the direct cause in many instances. That some poison is at work is certainly indicated by the degenerative changes in the kidneys and other internal organs sometimes found in fatal cases.

Symptoms.—The attack may commence suddenly or may be preceded for 12 or more hours by such manifestations as coated tongue, constipation, malaise, irritability, abdominal discomfort, and loss of appetite. Vomiting then begins, at first of the food ingested; later merely serous or mucous or finally bilious, or sometimes brownish or blood-stained. The vomiting is often forceful, with much retching, and occurs whenever anything

¹ Bull. soc. de pédiat., 1905, VII, 41.

² Amer. Journ. Med. Sci., 1903, CXXV, 629.

³ Amer. Journ. Dis. Child., 1912, III, 209.

⁴ Lancet, 1911, II, 8.

⁵ Ann. de méd. et de chir. inf., 1905, IX, 150.

⁶ Arch. de méd. des enf., 1905, VIII, 741.

⁷ Amer. Journ. Med. Sci., 1904, CXXVIII, 966.

whatever is swallowed, or even without this with varying frequency; sometimes in severe cases as often as every half hour. There is little or no elevation of temperature; often abdominal pain; and occasionally tenderness, obstinate constipation, urgent thirst, scaphoid abdomen or sometimes tympanites, and in some cases headache. The pulse is sometimes rapid and weak, sometimes slow or intermittent. As the attack continues the coated tongue becomes dry and brown; the prostration extreme; there is an anxious expression of face; the eyes are sunken; there is an odor of acetone on the breath; the urine is scanty and sometimes albumin and β -oxybutyric acid appear in it. Acetone is constantly present later in the attack and in some cases at the outset also.

Course and Prognosis.—The attack lasts 2 to 4 days and sometimes longer, and recovery from it is usually rapid. The bowels open of their own accord or yield to purgatives; appetite returns; vomiting grows rapidly less frequent, and in a few days no symptoms remain except the emaciation and loss of strength, which soon disappear. The prognosis is on the whole good. In spite of the extreme prostration not infrequently present, recovery generally takes place. This is not, however, without exception. I have reported¹ 2 very typical cases terminating fatally with evidences of nephritis, and I have seen another ending in death, and still another in which the great debility resulted in thrombosis of the artery of one leg with consequent gangrene and loss of the foot by amputation. A number of other fatal cases are on record in medical literature. There is always a recurrence of attacks at irregular, or occasionally somewhat regular, intervals usually of several months, and this may continue throughout several years, although as puberty is approached there is a tendency for the attacks to cease. In some instances, as in those reported by Rachford,² the attacks of vomiting have been replaced by migraine later in life.

Diagnosis.—This often presents many difficulties unless there is a history of previous attacks of a similar nature. Even with this it is important to make a most careful examination of the body, including the urine, to eliminate the presence of other causative conditions. The obstinate constipation may suggest *intestinal obstruction*, and I have seen cases in which the differential diagnosis was most difficult. There is seldom, however, severe abdominal pain in recurrent vomiting, and in the case of intussusception there are characteristic distinguishing symptoms. Yet cases showing typical symptoms of recurrent vomiting have been found at autopsy to depend upon obstruction of some sort, as at the pylorus (Russell)³ or in the duodenum (Gordon).⁴ *Appendicitis* usually has less severe vomiting and is attended by localized tenderness and a greater degree of pain, together with fever. Yet I have known of operation being performed on the theory that the vomiting depended upon a chronic appendicitis. *Acute indigestion* has often the history and the evidence of indiscretion in diet and the vomiting is of shorter duration. *Acute febrile diseases* with an onset with vomiting are generally soon distinguished by the development of other symptoms. *Tuberculous meningitis* or other serious disorder of the brain has, it is true, vomiting of a suggestive forceful character, but the nature of the other symptoms eventually eliminates recurrent vomiting from consideration. *Nephritis*

¹ Trans. Assoc. of Amer. Phys., 1900, XV, 16. Amer. Journ. Med. Sci., 1900, Nov.

² Archives of Ped., 1898, XV, 607.

³ Proc. Royal Acad. of Med., 1909-10. Dis. of Child., 78.

⁴ Brit. Med. Journ., 1906, II, 866.

may produce uremic attacks with severe vomiting. Careful examination of the urine will settle the question.

Treatment.—In cases where premonitory symptoms show themselves the attack may sometimes be aborted by stopping all food and by procuring a very free evacuation of the bowels by purgatives, such as Rochelle salts, citrate of magnesia, or calomel. Enemata do not answer, since it is not the mere unloading of the bowel which is desired, but the elimination of poisonous substances from the blood. If vomiting has commenced, a tentative trial of purgatives should be made, but this should not be persisted in if the medicine is rejected. No food or drug of any sort should then be given by the mouth. The child should be kept as quiet as possible in bed. To relieve the distressing thirst, small pieces of ice may be placed in the mouth, but this should be limited as much as possible, since the irritability of the stomach is very liable to be increased in this way. If the case is prolonged, rectal feeding may be tried; and where the character of the pulse, the appearance of the patient, or the scanty urination makes it evident that there is need of liquid in the tissues, enteroclysis may be given, or hypodermoclysis employed if necessary. The only form of medicinal treatment in my experience which offers much hope of checking the vomiting is the hypodermic administration of morphine in full doses. The relief is sometimes remarkably prompt and lasting, and I believe I have seen it without question save life. In other cases, however, it is not of much benefit. Bromides and chloral may be tried by the bowel, but are usually unavailing. As the attack subsides careful return to food may be made, employing such articles as broth, barley water, albumen water, and equal parts of skimmed milk and lime water.

Effort should be made in the intervals to prevent the recurrence of attacks. On the theory that the disease was an acidosis, Edsall¹ and Pearson² recommended keeping the child on full doses of bicarbonate of soda, $\frac{1}{4}$ to $\frac{1}{2}$ dram (0.97 to 1.94) 3 times a day, the amount given being increased greatly should any prodromal symptoms appear. I have known this treatment to appear very efficacious, but to fail in other instances. Care should be taken that the bowels are open daily, and at intervals of a week or two a freely acting purgative should be administered. Excitement and undue fatigue should be avoided. The diet should be digestible and simple, but of just what nature we cannot yet determine until the cause of the disease is better understood. Limiting the amount of fat and increasing that of the carbohydrate should certainly be tried, on the assumption that the disease may be due to an acidosis.

GASTRALGIA

Gastralgia, like vomiting, is a symptom of various conditions. In the broader sense of pain in the epigastrium, not necessarily arising in the stomach, it is common and may be the result of acute gastric indigestion, spinal caries with pain conducted along the nerves of the abdominal wall, malaria, renal colic, appendicitis, pneumonia, gastric ulcer, diaphragmatic pleurisy and other causes. In some children of delicate constitution or highly neurotic organization there may occur a true nervous gastralgia, apparently of a neuralgic nature, due to many diverse agencies, such as exposure, fatigue, and emotional disturbances. This is more common in

¹ *Loc. cit.*

² *Arch. of Pediat*, 1903, XX, 505.

childhood than in infancy. In the milder cases the pain is slight and of short duration; in the more severe it may be continuous and so intense that prostration and faintness are present and perforation may be suspected. In infancy symptoms may be produced which are apparently those of intestinal colic, but which are relieved by the eructation by the patient of gas from the stomach, thus indicating the true nature of the affection.

A careful study of the various possibilities should be made in order to reach a correct diagnosis of the form of gastralgia present. In the way of treatment, the cause must be sought for and removed if possible. In those cases in which the pain appears neuralgic, or due to an accumulation of gas in the stomach, treatment during the attack consists in confinement to bed; abstinence from food; hot applications to the abdomen, such as hot water bags, stupes, or mustard plasters; the administration of a carminative such as ginger, oil of cloves, best combined with spirits of chloroform, or with milk of asafetida or compound spirits of ether; and if necessary the giving of an opiate. Between the attacks efforts must be made to improve the general health and to correct any faults of diet. The best results are sometimes obtained by the administration of quinine or arsenic.

ANOREXIA

This is a symptom common, in a moderate degree, to a great number of diseased states, and is oftenest of brief duration. All acute febrile disturbances are liable to be accompanied by it, and all acute cases of gastric indigestion have it well-developed. Anorexia of a more chronic nature is dependent upon various causes. Some instances of chronic intestinal indigestion in older children exhibit it to a decided degree. In other cases a child is fed so often or so largely that it is impossible for the appetite to be good; there being no opportunity to grow hungry. At times the anorexia is in reality fictitious, appetite being in no way actually disturbed, but food being refused on account of pain produced by the effort to take it. This is seen in various disorders of the mouth and throat. There is a group of cases in which anorexia is by far the most prominent symptom, often being very persistent and giving the parents much anxiety. It is, namely, of frequent occurrence for an infant, who has previously taken food well, to empty but partially or entirely refuse one or more bottles daily, and to continue in this condition for weeks. There is no apparent alteration of any kind in the general health or spirits, except that constipation is a very common attendant. In some of these cases a constant error in diet may be discovered. In others no such cause can be found. Yet the condition is almost certainly a form of gastrointestinal indigestion of which the anorexia is the only apparent symptom.

In older children anorexia of this sort is even more common. Many such seem never to care about eating, and every meal-time is a struggle on the part of the parents to persuade the child to take food. A cause can often be discovered for the trouble. In some cases it is the absence of sufficient out-of-door life and exercise. In others small amounts of food are being frequently given to the child between meals, and this is often of an entirely unsuitable nature, such as sweetmeats, cake, and the like; and as a natural result the digestive processes are interfered with and anorexia follows. In certain cases it is associated with constipation. In still others the condition is truly constitutional, the child being from birth

a light eater without the normal desire for food. In some instances of this sort a very thorough assimilation of the food takes place, and the health does not suffer; but in others the child is always more anemic and less well-nourished than should be the case. The loss of health may be the result of the loss of appetite; but in many instances the anorexia is only one of the manifestations of the general delicate state of health which is characteristic of the patient, and which is in many cases inherited and often cannot be satisfactorily overcome. Finally, in certain cases presently to be described the anorexia may be purely nervous (*Anorexia Nervosa*).

Treatment.—The treatment of these cases of habitual anorexia is often most perplexing. It is primarily that of the cause when this can be discovered. Careful attention should be paid to the condition of the bowels and any tendency to constipation overcome. The hygiene of the patient must be considered and an out-of-door life prescribed. For older children cool morning baths may be employed. Massage may be of service and temporary change of climate, such as a visit to the seashore or the mountains, may be most efficacious. Regulation of the diet is most important. All eating between meals should be positively forbidden. An infant who refuses its bottle had better not be fed until the next usual hour for feeding arrives. In the acute cases the reduction by the child itself of the amount of food taken is probably Nature's own method of cure and should not be interfered with; and it is usually well to reduce temporarily the strength of the food given. Sometimes the omission for a time of the fat from the diet is of advantage; eggs and whole milk being forbidden; or solid food entirely withdrawn for a season. In some cases it is best to withdraw from the diet-list foods which are satisfying but not very nourishing. Thus children who have the appetite entirely satisfied by a bowl of clear broth, and who will eat nothing after it, should have no such food given.

As far as medication goes I have had the best results from the administration of an alkaline bitter tonic before meals, such as the combination of tincture of *nux vomica*, tincture of gentian and bicarbonate of soda, in doses suitable to the age. Occasionally a mineral acid is more efficacious given after meals. The administration of sherry, port wine, or whiskey 3 times a day is often of benefit, care being taken in older children that no fondness for alcohol is developed. The addition of a bitter tonic to the stimulant tends to prevent this. With the tonic treatment should be combined purgation every 5 to 7 days, with the intention of removing toxic material from the circulation. This is in addition to the daily regulation of the bowels referred to.

Anorexia Nervosa.—This form of loss of appetite must receive a short separate consideration. It is always of purely nervous origin and is in some instances a hysterical manifestation. The disease as occurring in children has been ably reviewed by Forchheimer.¹ The simplest form is that developing under the influence of emotional excitement. Pleasurable anticipation, as of a visit to the theater or the circus, may remove for a time all desire for food. The excitement, for instance, of the first going to school, or the influence of anxiety or grief, may diminish or abolish the appetite for days or longer. It is the more chronic cases, however, which give trouble to the physician. In this category belong some of the instances already referred to of persistently poor appetite in the period of childhood. The repugnance to food may be so great

¹ *Archiv. of Pediat.*, 1907, XXIV, 801.

that insistence upon eating beyond a certain amount results in the child vomiting what it has taken. An element of hysteria plays a large part in many of the cases. A child who has acquired a reputation for having no appetite, subconsciously feels obliged to live up to this. Consequently the anxious urging of food upon such a subject, or the remarks made in his presence by the parents regarding the lack of appetite, tend to fix firmly in the child's mind the unwillingness to eat. I have previously reported cases of this nature.¹ Imitation, too, plays an important rôle, and if other members of the family eat but little and openly express dislike for certain articles of diet the nervously susceptible child is very liable to do the same thing. In older children this hysterical anorexia may become a very serious menace to health (Forchheimer),² but even in infancy the influence of the idea upon the appetite is much more frequent than ordinarily supposed and may reach a threatening degree. In a case previously reported³ an infant of 21 months refused all nourishment but breast-milk, which had become insufficient. After a month of effort gavage 3 times a day was ordered and was required for a period of 6 months. This anorexia apparently depended upon fear of anything given from a spoon or glass, the fear dating from an attack of illness at 9 months, when medicine had to be administered in this way.

Treatment.—The treatment of anorexia nervosa depends upon the case and requires careful study by the physician and coöperation by the parents. Certainly all anxious discussion of the food in the presence of the patient must be abandoned and little apparent notice taken of whether the child eats or not. A complete alteration of diet may be of benefit. Change of air is often useful and still more is change of scene. Sometimes the temporary separation of the child from the family is the most efficacious method of treatment. In cases where the health is suffering and other means are insufficient, gavage may be necessary for a time.

STENOSIS OF THE PYLORUS

(Hypertrophic Pyloric Stenosis; Pylorospasm)

An instance of this condition, the earliest reported, was by Hezekiah Beardsley,⁴ although its nature was not recognized at the time. Possibly a still earlier case is that described by Armstrong⁵ in 1777 as "spasm of the pylorus." At least the autopsy revealed this condition, although the case is described as one of "watery gripes." A case in infancy was published by Williams⁶ in 1841, and another by Siemon-Dawosky⁷ in 1842. Landerer⁸ in 1879 and Maier⁹ in 1885 described pyloric stenosis as found at autopsy in adults. No further notice was taken of the subject until the contribution of Hirschsprung¹⁰ in 1888. It is only in recent years that the disease has become the subject of much interest, and has been discussed in very many contributions to medical literature. Undoubtedly it is of

¹ New York Med. Jour., 1914, June 6.

² *Loc. cit.*

³ Arch. of Pediat., 1908, XXV, 321.

⁴ Cases and Observations by the Medical Society of New Haven County in the State of Connecticut, 1788; republished by Osler, Arch. of Ped., 1903, XX, 355.

⁵ An Account of the Diseases Most Incident to Children.

⁶ Lond. and Edin. Month. Journ. of Med. Sci., 1841, 23.

⁷ Caspar's Wochenschr. f. die gesammte Heilkunde, 1842, 105.

⁸ Inaug. Dissert., Freiburg, 1879.

⁹ Virchow's Archiv., 1885, CII, 413.

¹⁰ Jahrb. f. Kinderheilk., 1888, XXVIII, 61.

much more frequent occurrence than ordinarily supposed. Ibrahim¹ in 1910 states that up to the period of writing 598 cases of stenosis had been recorded. Numbers of cases have since then been observed, to such an extent that physicians have ceased reporting them. I have, for instance, observed 6 cases within a little over 1 month.

Pathogenesis.—The nature and origin of the malady is even yet not entirely clear. There appear to be certainly two factors in producing stenosis, and consequently two classes of cases, not however, sharply distinguishable: (1) *pylorospasm*, dependent upon a spasm of the muscular layer of the pylorus; (2) congenital hypertrophy of all the tissues, but especially the muscular fibres, viz. *hypertrophic stenosis of the pylorus*. The first element predominates in some cases, the second in others. Perhaps a third factor may be not without influence in completing stenosis, viz. a swelling of the mucous membrane of the pylorus. This is supported by the observations of Weill and Pehu² who state that examination of the pylorus in cases reported by them showed cellular infiltration resulting from inflammation. In the majority of the instances, it is probable that a certain degree of hypertrophic stenosis is present, but that the stomach was at first able to overcome this; and that finally a large element of spasm develops in addition and closes the pylorus, while at the same time the expelling power of the stomach diminishes. In other cases it is possible that spasm is the sole or principal cause. The numerous instances of recovery without operation show the etiological importance of spasm, the contraction relaxing and the muscle of the stomach regaining its normal tone. That spasm may exist alone is indicated by the fact that infants have exhibited typical symptoms of stenosis, died suddenly from an intercurrent malady, and at autopsy disclosed no pyloric narrowing whatever. On the other hand the findings at operation and at autopsy demonstrate in most instances the reality of actual organic changes in the pylorus. That some degree of organic change may be present even in the cases which recover, or in those which have exhibited no symptoms, has been shown by the discovery of hypertrophy of the pyloric tissues in individuals dying from other causes. It would seem, indeed, very probable that more or less hypertrophy is present in every case of stenosis, even when by itself it is not sufficient to produce the symptoms.

As to the method of production of either spasm or hypertrophy, there is still nothing certain. Thomson³ believed that the spasm is primary, due to the irritation from intrauterine swallowing of the liquor amnii, and that hypertrophy follows this. Some views would make the hypertrophy secondary to spasm from other prenatal causes; and others secondary to some irritation of the duodenum and stomach, possibly a hyperacidity. Still other investigators maintain that the hypertrophy is a primary fetal development and that spasm is secondary and produced by this, and this would appear to be the most probable explanation.

Etiology.—The disease shows itself in the first weeks or even in the first days of life. A quarter of the cases occur in the first 4 days and an equal number from this time up to 2 weeks of age (Pfaundler).⁴ Very many more males are affected than females. The majority of cases reported have been observed in breast-fed infants but this may per-

¹ Münchener med. Wochenschr., 1910, LVII, 1154.

² Arch. de méd. des enf., 1910, XIII, 507.

³ Scott. Med. and Surg. Journ., 1897, I, 511.

⁴ Pfaundler and Schlossmann, Handbuch der Kinderheilk., 1906, II, 1, 183.

haps be an accidental occurrence. A family influence is exceptionally noted, in that more than one infant of the same parents has been attacked, or that the parents have suffered from digestive diseases. Disturbances of digestion in the infant, including that resulting from overfeeding or from excessive acidity, are among the assigned causes of spasm; or possibly reflex influences arising in distant parts of the body, a congenitally sensitive gastric mucous membrane, or a neuropathic constitution may be active in producing it.

Pathological Anatomy.—In the cases exhibiting alterations at autopsy the pylorus is found elongated, thickened, with the stiffness of cartilage, and with the mucous membrane projecting into the duodenum, thus bearing a resemblance to the appearance of the uterine cervix in the vagina. Section of the pylorus shows great thickening of the walls, and the lumen occluded by this and by the swelling of the longitudinal

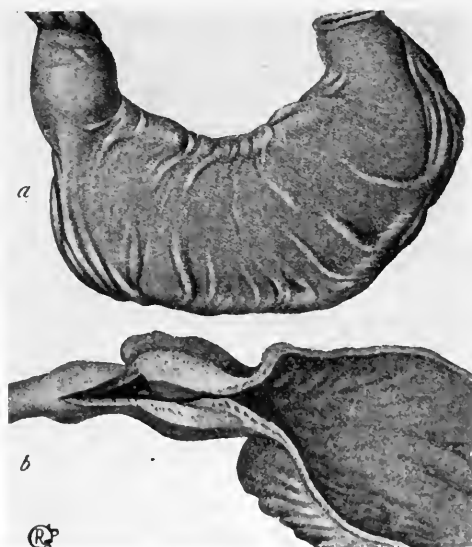


FIG. 241.—HYPERTROPHIC PYLORIC STENOSIS IN A SIX WEEKS OLD INFANT.

a, Tumor; b, longitudinal section, showing hypertrophy of circular fibers and extremely narrow lumen. (*Pisek and LeWald, Arch. of Pediat., 1912, XXIX, 911.*)

folds of the mucous membrane (Fig. 241). The appearance is much the same whether due to spasm or to hypertrophy, and only careful study, including a slow stretching of the pylorus and microscopical examination, can show which condition is the principal cause of the stricture. If the organic change is the predominant one there is found evident hypertrophy of the muscular layer, especially of the circular fibres. Increase of the longitudinal muscular fibres and of the thickness of the mucous and subcutaneous layers is present to a less degree. The stomach may be contracted or both it and the esophagus dilated.

Symptoms.—Two classes of cases have been described: viz. pyloro-spasm and hypertrophic stenosis of the pylorus; but clinically the symptoms differ only in degree and may be considered together, especially since a combination of the two factors is present in probably all cases. Evidences of the disease may appear in the first days of life. This is particularly true of cases depending chiefly upon very decided hyper-

trophic stenosis. In such instances an apparently perfectly healthy and well-developed infant a few days old begins to *vomit* without cause. In the majority of cases the symptoms are longer delayed and appear only after the child has been well for several weeks; and the vomiting then perhaps begins immediately consecutive to a disturbance of digestion. In these latter cases spasm is probably an important factor. In either event vomiting is at first entirely uncharacteristic and only occasional. Steadily, however, it grows more frequent, taking place after every ingestion of food, either very promptly or after a delay of some hours, all or only a portion of the food being ejected. If *dilatation* of the stomach with retention of the contents develops, as is the case in many instances, the vomiting may be of a characteristic projectile character and expel at times much more food than had been taken at the last feeding; this showing a tendency for food to accumulate in the stomach. The empty-



FIG. 242.—STENOSIS OF THE PYLORUS.

Boy of $1\frac{1}{2}$ months, in the Children's Hospital of Philadelphia. Illustration shows two peristaltic waves. General condition very poor. Rammstedt operation, but death occurred 20 days later.

ing of the organ in these cases may occur at much longer intervals and perhaps only once or twice daily, the whole of the food taken since the last vomiting being ejected at one time. The loss of gastric motor power is shown by the evidences of dilatation and by the finding with the stomach tube or by examination with the x-ray of food still present in the stomach from 4 to 10 hours after it had been ingested. The food vomited is more or less altered, depending upon the time it has remained in the stomach, and there is often an admixture of mucus and sometimes of streaks of blood. Bile is rarely regurgitated and there is no evident nausea. There is sometimes an increase of hydrochloric acid in the vomited matter, in other cases not.

Constipation is a very characteristic symptom. There may be no fecal stool for some days if the stenosis is complete, what is passed being not feces, but merely dark-brown or dark-green mucus. If the stenosis is intermittent, or not complete, the stools may be fecal. *Loss of weight* is steady and rapid in most cases and an extremely emaciated state may

be reached finally; the abdomen being sunken, except the epigastrium which is distended. Yet to this there are occasional exceptions and the weight may remain almost stationary even in cases where there is no discoverable evidence that any food reaches the intestine. *Peristaltic waves* in the stomach, visible through the thin abdominal walls, constitute a very characteristic symptom, present in nearly all cases. They are seen especially after food has been taken, or just before vomiting is about to take place, but may occur at any time, even during sleep, or may be brought into evidence by stimulation of the organ through friction of the abdominal walls. They start upon the left side, below the costal margin, and pass toward the pylorus, from one to two being visible at a time and suggesting the presence of large balls rolling under the abdominal walls (Fig. 242). The peristalsis does not appear to be productive of pain. *Pain*, however, sometimes seems to follow the ingestion of food, and an infant may begin to take nourishment with avidity, but stop with a cry or with evidences of distress after a few mouthfuls. A *pyloric tumor* may be felt in a large proportion of cases. It is situated slightly to the right of the middle line, under the edge of the liver, or often lower near the vertebral column and toward the umbilicus, is movable, and gives the sensation of a small, hard body of the size of a small nut or an enlarged lymphatic gland about 1 to 1.5 inches (2.54 to 3.8 cm.) in length. Slight anesthesia may be necessary to make the examination satisfactorily. The urine is high-colored and scanty, as a result of the inability to retain and absorb liquids. There is no fever unless from a complicating digestive or other disturbance.

Course and Prognosis.—Concerning the probability of recovery in stenosis of the pylorus there is much difference of opinion. The chances are greater in those cases in which the development of symptoms is delayed. Undoubtedly many recover when spasm is the chief element. Heubner¹ has reported 19 recoveries in 21 cases treated medically. In such cases the vomiting may gradually diminish, fecal matter appear in the stools, and the excessive peristalsis of the stomach finally disappear. Patients with a high degree of hypertrophic stenosis, on the other hand, grow rapidly worse, the symptoms continuing until the infant is extremely wasted, and death finally taking place after several weeks of illness, unless timely operation has been performed. Even after operation the mortality has been high. Ibrahim² found that in the 172 reported cases of operation the mortality had been 50.6 per cent. In the medically treated cases it was 36.5 per cent. The total mortality in the 598 cases of both kinds of stenosis equalled about 40 per cent. Could a positive differential diagnosis of the two classes of cases be made promptly, and those with predominant hypertrophy be operated on at once, the mortality figures would undoubtedly be much reduced, since, if operation is to be done, the earlier it is performed the better is the chance for the patient. A more prompt decision, earlier resort to operation, and improved technic have of recent years greatly increased the percentage of recoveries; as, for instance, in Richter's³ experience of but 13.6 per cent. mortality after operation, and Strauss⁴ of but 4.6 per cent. All statistics are, however, to a certain degree misleading. It is not the relative mortality of medical and surgical treat-

¹ Therap. d. Gegenwart, 1906, N. S., VIII, 433.

² *Loc. cit.*

³ Journ. Amer. Med. Assoc., 1914, LXII, 353.

⁴ Journ. Amer. Med. Assoc., 1918, LXXI, 807.

ment which is the important matter, but the question of the sort of treatment required for the individual case.

Diagnosis.—Mistakes are easily made because the symptoms are not always typical and because, too, the possibility of the existence of stenosis does not occur to the physician's mind. The chief characteristic symptoms are the early development; the obstinate forcible expulsive vomiting without sufficient cause in the way of digestive disturbance; the characteristic peristalsis; constipation; and, if discovered, the pyloric tumor. Congenital conditions such as atresia of the pylorus or stricture of the duodenum may give rise to mistakes, but both of these malformations are extremely uncommon; and the latter exhibits biliary vomiting while the former is the cause of death in a very few days. A mistaken diagnosis may also arise in other ways. Thus, for instance, the stenosis can occur from without, as by pressure of the cecum, as in a case described by Toporski.¹ In another instance (Gittings)² an appearance of what seemed to be gastric peristalsis was in reality produced by two folds of the colon overlying the stomach; and Hoffa³ has reported a number of cases in which gastric peristalsis was present without the slightest reason to suspect stenosis. In stenosis of the esophagus the food is regurgitated entirely unchanged, almost immediately after attempts at swallowing are made; and even if dilatation of the esophagus is present and vomiting is somewhat delayed, there is no evidence of gastric digestion in the ejected matter. There is also no discoverable gastric peristalsis or pyloric tumor. Valuable for diagnostic purposes is the employment of the Röntgen ray after the administration of bismuth. This will show a great retardation, or even entire failure of passage of the bismuth from the stomach into the duodenum. It is not, however, conclusive, as I have seen the retardation combined with projectile vomiting occur in a case proven by autopsy to be chronic gastritis. It is also usually unnecessary inasmuch as delay in the emptying of the stomach can be determined equally well with the stomach-tube.

The distinction between the two classes of cases is usually not possible with certainty, since the two conditions are probably always associated, although in varying degrees. In the cases in which pylorospasm predominates vomiting often begins later and is more irregular and with longer intervals; the obstruction is not so complete, as shown by the character of the stools; the wasting is not so rapid; and dyspeptic disturbances are more liable to be present, and nervous symptoms may exist. In cases where hypertrophic stenosis is the principal factor, the reverse of all this is true and the symptoms appear earlier and are in every way worse. Yet, on the other hand, all the milder symptoms with the presence of fecal stools may exist in cases in which autopsy shows that decided hypertrophy had been present. It is to be borne in mind that hypertrophic stenosis may exist and remain latent indefinitely; may be recognized only at autopsy; or may show symptoms only after years. It is possible, too, for what appears to be simple spasm of the pylorus to show itself for the first time in childhood, and not in infancy. A case of this nature was reported by Graham.⁴

Treatment.—Inasmuch as the diagnosis from an ordinary digestive disturbance is not at first possible, the earliest treatment must be purely

¹ *Jahrb. f. Kinderheilk.*, 1910, LXXII, 285.

² *Arch. of Pediat.*, 1911, XXVIII, 661.

³ *Monatsschr. f. Kinderheilk., Orig.*, 1912, X, 523.

⁴ *Amer. Journ. Dis. Child.*, 1911, II, 407.

symptomatic. Even if it be recognized that stenosis is present the same holds true, and a thorough trial of medical treatment should be instituted. Efforts must be made to relieve the vomiting as far as this is possible. Lavage of the stomach, practised once or twice daily, is the best means for this purpose, using a warm normal salt solution, or a 1 per cent. solution of bicarbonate of soda. If much exhaustion follows the procedure must be abandoned. Small rectal enemata of normal salt solution should be given several times daily to supply the liquid needed by the system. Warm baths serve the same purpose to a certain extent and tend to favor relaxation of the spasm, and hot, wet applications to the gastric region are useful in the same way. Dietetic treatment is very important. Breast-milk should be obtained when possible in cases of artificially fed children who have shown any symptoms of digestive disturbance. If this is not feasible, the fat of the diet should be reduced decidedly. If the child is already nursing, it should not be weaned, but the milk may be given diluted or in smaller quantity and at longer intervals. In some instances albumen water or whey made from skimmed milk answers well; if not, other food must be experimented with. Very little food of any sort should be given at any one time, and the intervals may be long or short according to the results which trial shows to be the best. Immediately after lavage is a suitable time for one of the feedings. In some cases feeding by gavage has answered better than any other method. Food given by the bowel is usually of little value, but albumen water or other nourishment may be tried in this way.

Treatment by drugs is generally of little avail, but the opiates, bromides or belladonna may be tried in the hope of relieving spasm and lessening the gastric irritability. Belladonna certainly seems of value in some cases.

After a fair trial without avail of the methods mentioned, the question of operative interference arises. This is a serious problem. It is important not to resort to it too soon, but it is equally important not to delay it so long that the infant has grown too weak to tolerate any surgical intervention. It is a radical mistake, in my opinion, to regard every case of stenosis as a subject for operative interference; but continued experience with this disease has convinced me, on the other hand, that the most common therapeutic error is a too prolonged delay before operation is resorted to. I have had cause for self-reproach for undue delay in advising operation, but have not yet regretted recommending an early operative interference. Each case must be a rule for itself, and the general condition of the infant, and the gain or loss of weight under observation must be the guide as to the length of time which may be allotted for medical treatment; but after a careful and thorough trial of this for not more than 1 or 2 weeks at the outside, without change in the general condition, nothing is to be gained and much lost by persisting with it. Should the infant be in bad condition when first seen, or improvement does not take place promptly, the time mentioned is too long, and the operation should be proceeded with immediately or perhaps after a day's abstinence from food, together with the vigorous employment of enteroclysis and hypodermoclysis. Several operations have been proposed, that of gastroenterostomy appearing to have a better mortality record than either divulsion or pyloroplasty. More recently the Rammstedt¹ operation of simple splitting of the hypertrophied mucous membrane has given such favorable results that it is now the one most commonly

¹ Med. Klinik., 1912, VIII, 1702.

employed. It possesses the great advantage that a skillful operator requires not more than 20 minutes for its completion. A somewhat similar operation has been devised by A. Strauss¹ in which the muscular layer is split, and then again divided to form flaps over the exposed mucous membrane. Strauss reports but 3 deaths out of 65 cases. After operation feeding may be commenced almost immediately. Breast-milk should be used if possible, but only very small amounts given at first, and the full quantity permitted only by a week or more after the operation. A half-ounce (15) or less every 2 hours may be given at first, and this increased in the course of 2 or 3 days to 1 oz. (30) every 3 hours. Sometimes it is better at the beginning to dilute with one-quarter water or lime water. The breast-milk should at first be given from a bottle. When convalescence is well under way, in about a week, nursing directly from the breast may be resumed. If this is found not to agree well, administration of the food from the bottle should be promptly recommenced. Vomiting is liable to occur during the first few days, but the keeping the infant's head somewhat elevated tends to check this. In many cases the employment of hypodermoclysis once or twice a day is of great service when a sufficient amount of liquid cannot be taken or retained.

CARDIOSPASM

This condition occurring in infancy is analogous to the pylorospasm of that period of life. With propriety it might be described as a form of spasm of the esophagus. Attention has been called to it by Freund,² Göppert,³ Beck⁴ and others. The disease is probably more frequent than ordinarily supposed. It is one of the possible causes of uncontrollable vomiting in early childhood or infancy. The pathogenesis of cardiospasm is not well understood. The principal *symptom* is the prompt vomiting after the first swallowing of food. This act is attended by very characteristic actions, viz. regurgitation and repeated reswallowings; distinguished, however, from rumination in that there is no difficulty at all attending deglutition in the latter disease. The passage of a sound encounters obstruction at the cardiac orifice of the stomach. The entrance of food through a small catheter passed directly into the stomach is not followed by vomiting. In the few cases as yet reported the prognosis appears good. In the line of *treatment* a sound may be passed systematically, or the stomach may be allowed to rest entirely for a few days while rectal alimentation is employed.

DILATATION OF THE STOMACH

A moderate dilatation of the stomach is of common occurrence especially in infants. A degree of it which is of sufficient importance to produce symptoms and demand special treatment is not frequent.

Etiology.—Dilatation of a *chronic* nature may follow a mechanical obstruction such as stenosis of the pylorus, but the most frequent cause is a loss of tone of the muscular walls dependent upon constitutional conditions, notably rickets and infantile atrophy, combined with constant overfeeding especially with artificial food, resulting in indigestion, decomposition and formation of gas, and the accumulation of this and dis-

¹ *Loc. cit.*

² Monatsschr. f. Kinderh., 1903, II, 15.

³ Therap. Monatsschr., 1908, XXII, 390.

⁴ Monatsschr. f. Kinderh., Orig., 1911, IX, 555.

tention of the stomach. There may be observed an *acute* dilatation of the stomach, and of the intestine as well, occurring sometimes in the course of respiratory disease or after abdominal operations; and very rarely a sudden and even fatal acute dilatation may develop in infants without any discoverable cause. The disease in this form has been reviewed by Lucas.¹

Pathological Anatomy.—The earliest changes are simple relaxation of the gastric walls, but this is followed in the chronic cases by atrophy of all the layers. The shape of the stomach becomes much altered, the lesser curvature changing little while the greater extends downward much farther than normal, the dilatation being most marked near the cardia. This produces a large pouch, and the normal shape and position are thus entirely altered. The gastric capacity is increased many times above that normal for the age. With the dilatation there may be combined a gastropotosis, and there is generally an accompanying dilatation of the intestine.

Symptoms.—These are principally those of chronic gastric indigestion. (See p. 723.) There is often vomiting after meals or at irregular times, eructation of gas, and abdominal discomfort. The vomiting is generally not frequent, and the amount ejected is surprisingly large. The appetite is sometimes lost, sometimes abnormally great. The general health steadily fails and the child becomes anemic and emaciated. Constipation may alternate with diarrhea. On physical examination the gastric region is found distended and tympanitic, and sometimes the stomach can be clearly outlined by percussion. A succussion-sound may be elicited in some instances.

Prognosis.—The outlook is unfavorable when the disease is due to pyloric stenosis. When dependent upon other causes the prognosis is usually good, if dilatation is not excessive, although recovery may be tedious. If due to indigestion and overfeeding, recovery should follow without difficulty; when rickets is the predisposing factor a cure will not be obtained until the constitutional trouble is removed. The development of gastric dilatation may constitute a dangerous complication of other diseases, especially pneumonia.

Diagnosis.—It is very easy to mistake a distended colon for a distended stomach, and when both viscera are dilated distinction by simple percussion is almost impossible. To determine the matter the stomach may be filled with water a few hours after a meal and the lower limit of percussion dullness now sought for. If this nearly reaches the transverse umbilical line the organ is dilated. The administration of bismuth followed by examination with the Röntgen ray may likewise be used to outline the lower gastric border.

Treatment.—Correction of the diet is essential. The food should be nutritious and unirritating; suitable to the age and the digestive power of the patient. It must be given frequently and only in small quantities, the amount of fluid especially being reduced in older children. When the appetite is inordinate, not sufficient food should be allowed to satisfy it. As the stomach grows smaller the excessive hunger will disappear. In general those articles of diet are to be avoided which tend to produce an accumulation of gas in the stomach. Systematic lavage daily is of value in cases where dilatation is decided. Every care must be taken to improve the general health by tonic and hygienic measures. Massage is of service and the administration of strychnine is of value. In cases

¹ Arch. of Pediat., 1909, XXVI, 151.

of acute dilatation the stomach-tube should be passed at once and a hypodermic injection of eserine given.

GASTRIC HEMORRHAGE

Etiology.—This is a symptom produced by a variety of causes. It has already been referred to under *Melena neonatorum* (p. 266). It is also one of the manifestations of a more general tendency to hemorrhage seen in the hemorrhagic disease of the new born already described (p. 264), and may occur in scurvy and in some forms of purpura. Apart from the ulcers occasionally found in melena, gastric hemorrhage may result at a later period from ulceration of the stomach or duodenum, or from injury by a foreign body.

Symptoms.—These consist solely in the vomiting of blood which, on investigation, is shown to have its origin in the stomach. Hemorrhage supposed at first to arise in the stomach may come from the nose, mouth, or lungs, the blood having been swallowed and then vomited. Blood from the lungs is bright-red in color, coughed up, and frothy if it is expectorated directly without previous swallowing. If the hemorrhage from the stomach is free, the blood may be bright-red; if it has taken place slowly or if it has been lying for some time in the stomach it is dark-brown or black in color. The **prognosis** is usually serious, since the cause is generally a severe one.

Treatment.—This depends to a certain extent upon the cause, but is in general symptomatic. The patient should be at absolute rest, an ice-bag placed over the stomach and small pieces of ice swallowed. No food at all should be given. An opiate hypodermically is of benefit to check gastric peristalsis. Stimulants may be required by way of the skin or the rectum. In the line of medicinal treatment suprarenal extract in large doses is the best remedy. Gelatine internally is sometimes of service also.

GASTRIC ULCER

Etiology and Pathological Anatomy.—This is an unusual condition in early life. Stowell¹ collected 35 published cases, including 1 of his own; 10 of these, however, being in subjects over 12 years of age. Jacobi² added a number of others to the list, and Lockwood³ has collected about 125 cases, including a number of unpublished instances. Several causes may produce it in early life. It is oftenest seen in melena, although hemorrhage from the stomach from this disease may occur without ulceration discoverable at autopsy. Sepsis in the new born is likewise a cause; acute gastritis, especially that from corrosive poisoning occasionally produces it; it may exceptionally be found in chronic gastritis, and in rare instances tuberculous ulceration of the stomach is found.

The primary round peptic ulcer of the stomach is rare. It is only toward the end of later childhood approaching puberty that it begins to be comparatively more frequent. It is more common in girls than in boys. The peptic ulcer is usually single, and has the pathological characteristics of the lesion as seen in adult life with its tendency to perforation. This is in contradistinction to gastric ulcers from other causes mentioned, which are more often multiple and sometimes very numerous,

¹ Med. Rec., 1905, LXVIII, 52.

² New York Med. Journ., 1909, XC, 837.

³ Surgery, Gynec., and Obstet., 1914, XIX, 462.

and frequently have the characteristics of erosion merely or of follicular ulceration.

Symptoms.—These are very often not typical, simulating those of acute gastritis, which indeed may have ulceration accompanying it; and it may be that the lesion is discovered only at autopsy. Sometimes the perforation of an ulcer and subsequent peritonitis is the first indication that any serious trouble has existed. In other less frequent cases there are the characteristic symptoms of vomiting of blood and the passage of reddish or black stools, with pain and tenderness in the gastric region. The pain is sometimes referred to other parts of the abdomen and the existence of appendicitis may be suspected. The **prognosis** is serious in all forms of ulcer in which the symptoms are sufficient to render a diagnosis even provisional.

Treatment.—The measures to be employed consist in the application of cold to the region of the stomach; absolute rest in bed; nourishment by nutrient enemata; and abstinence from all food and drink by the mouth, except that older children may swallow small pieces of ice. Morphine may be given hypodermically, and if the hemorrhage is severe suprarenal extract may be administered by the mouth and gelatine subcutaneously, with great care that the gelatine solution is properly prepared and sterilized. Later bismuth or nitrate of silver may be given by the mouth. If there is reason to believe that perforation has occurred, or if hemorrhage is uncontrollable, operation is indicated.

RUMINATION

(*Merycismus*).

Of this affection not many instances have been recorded in early life, although it is very probably more common than this would indicate. It appears to be analogous to the rumination normally occurring in some of the mammalia, but is in the human race a pathological process. The condition in infancy, at which period it seems more frequent than in childhood, has been studied by Aschenheim,¹ Brüning² and Schippers.³ The latter collected 12 published cases, including 2 of his own. Grulee⁴ adds 2 to the list, besides a number observed by himself.

The **symptoms** consist in a series of repeated regurgitations of small amounts of the food taken, occurring some time after its ingestion. Some of this is promptly swallowed again, some may be lost from the mouth. There is none of the forcible character which pertains to true vomiting. The nature of the food seems to be a matter of no influence. The prognosis, according to Grulee, is grave, fully 25 per cent. of the cases ending fatally. The administration of the bromides appeared to be of service in a case reported by Lust.⁵

FOREIGN BODIES IN THE STOMACH

As with the esophagus (p. 695), foreign bodies of very varied kinds may find their way into the stomach of infants or young children and, less frequently, of older children. From this organ they may be vomited, or may pass into the intestine and be voided. The **symptoms** are very

¹ Zeitschr. f. Kinderheilk., Orig., 1913, VIII, 161.

² Arch. f. Kinderh., 1913, LX, 116.

³ Nederl. Tydschr. v. Geneesk., 1914, I, 785.

⁴ Amer. Journ. Dis. Child., 1917, XIV, 210.

⁵ Monatsschr. f. Kinderheilk., Orig., 1911-12, X, 316.

indefinite. There may be an attack of choking at the time of swallowing, and possibly pain in the throat, or slight hemorrhage if the object is not of a smooth nature. When once in the stomach there are usually no symptoms whatever, unless irritation of the mucous membrane is set up by an object of a sharp or rough character. While in the viscus the body may be the cause of obstruction, inflammation, abscess, or perforation. It is important to remember in the matter of diagnosis that parents are frequently positive that a foreign body has been swallowed by the child, but that later this is found perhaps upon the floor. The employment of the Röntgen ray is, of course, necessary to establish the presence of the body. How long it may remain in the stomach without the production of symptoms and without danger depends much upon its shape and character. I recall an instance where the radiograms showed a metal whistle from a rubber toy remaining for 9 days in the stomach before it entered the pylorus.

Regarding **treatment**, the removal of the foreign body is necessary, but there need be no undue hurry to resort to such a serious operation as gastrostomy so long as there are no symptoms present, and a smooth body of moderate size is shown by the fluoroscope to be changing its position from time to time in the stomach: *i.e.* is not imbedding itself in the mucous membrane. The chance of passage through the pylorus is to be estimated by comparing the known size of the foreign body with that of the pylorus at certain ages. According to Hess¹ the diameter of the pylorus in the new born is 4.2 mm. (0.17 in.); at 2 to 3 months 6 mm. (0.24 in.); and at 6 months 7 mm. (0.28 in.); while Pfaundler² gives the circumference in the new born as 2 cm. (0.78 in.); and at 8 years 4 cm. (1.57 in.). Nevertheless I have known objects, after a considerable delay, safely to pass a pylorus the estimated size of which had seemed entirely too small to permit of this. Before any thought of operation is entertained, efforts should be made to aid the exit of the body and to protect the gastric mucous membrane. This is best accomplished by the administration of food which will coat the object, such as potato, bread, and cereal porridges. No purgative or emetic should be given.

A form of foreign body sometimes found is the **hair-ball**. This has occurred in children who are in the habit of chewing hair from the head, fur, wool from blankets, cotton, and the like. Generally the amount swallowed is not large and the material passes promptly into the intestine and is voided; but in some cases this does not occur and the size of the hair-ball is gradually increased by the continuance of the bad habit, until it may reach such dimensions that it forms a tumor recognizable by palpation and giving a soft, crackling sensation to the fingers. The passage of the stomach-tube may be interfered with by the presence of the mass. Possibly small amounts of hair are from time to time evacuated in the stools. The symptoms are indefinite, and consist at the most of moderate evidences of indigestion or of gastric distress. Operation is necessary.

MALFORMATIONS, MALPOSITIONS AND NEOPLASMS OF THE STOMACH

Malformations, with the exception of pyloric stenosis, are of great rarity. Malpositions are occasionally seen. The stomach may occupy the right side of the abdomen in cases of transposition of the viscera, or

¹ Amer. Journ. Child. Dis., 1914, VII, 184.

² Bibliotheca medica, 1898, H. 5, 35.

may be found partially within the thoracic cavity in diaphragmatic hernia. Through the presence of adhesions it may retain the more vertical prenatal position. It may be partially divided into two portions by a constricting wall; may remain of very small size, with thickened walls; or may exhibit complete closure at either orifice.

Morbid growths, too, are very uncommon in early life. Even tuberculosis of the stomach is rare. Osler and McCrae¹ collected 6 reported cases of cancer under the age of 10 years. Sarcoma and lymphadenoma are even less often seen. Other cases of morbid growths have since been reported.

ACUTE GASTRIC INDIGESTION

Although pathologically this is to be distinguished from acute gastritis, being merely a functional disturbance, the symptoms, especially at the beginning, are very similar, and a diagnosis cannot always be made. The two diseases are, however, different and should be considered separately. A distinction is also to be drawn between gastric indigestion in infants and that in older children.

Etiology.—The cause in infants, whether breast-fed or artificially fed, is the ingestion of unsuitable or of too large a quantity of milk. Sometimes the condition of the mother, as, for instance, after emotional excitement, produces milk which causes acute indigestion in the child. The giving the infant other articles of food to which it is unaccustomed may produce the same result. In older children overfeeding, or the eating of distinctly indigestible substances, such as cake, candy, pastry, unripe fruit, or even ripe fruit of certain sorts, and the like, has the same effect. Nervous influences are also a powerful factor, and acute indigestion may follow the fatigue from overexercise, excitement of any sort, and unusually hot weather.

Pathological Anatomy.—Probably no organic lesions exist, the disturbance being purely a functional one. The proper secretion of the stomach is interfered with and the motor power temporarily impaired.

Symptoms.—As a result of the disturbance of the gastric function the food lies too long in the stomach and undergoes abnormal changes. The first symptom is nausea, complained of by older children, and in infants shown by pallor of the whole face or of the region about the mouth and by perspiration of the forehead. Pain also may be present. Vomiting then occurs and the child is temporarily relieved. The vomited matter shows a distinctly abnormal quality. In infancy it is always sour and there are often large, curdy masses ejected. In older children it is also sour and various articles of food are found, showing little evidence of any digestive process having taken place even 5 or more hours after eating. A single act of vomiting may be sufficient to empty the stomach, but more often it is repeated after an interval; this occurring perhaps several times. Meanwhile there is often more or less fever, coating of the tongue, offensive breath, thirst, prostration, epigastric distress and pain, perhaps headache, constipation, and very frequently eventually diarrhea. The urine is scanty and high colored; the patient may be somnolent or very restless, or even convulsions may occur. In exceptional cases coma may follow. In the intervals between the attacks of vomiting there may be a desire for food, or entire loss of appetite may continue.

¹ New York Med. Journ., 1900, LXXI, 581.

Course and Prognosis.—If properly treated, and especially if no food is given, the acute symptoms are usually over in from 6 to 12 hours and all evidences of the disease in from 2 to 3 days. The prognosis is nearly always good, even though the symptoms may for a time seem alarming. Death, however, may take place especially in weakly subjects in early infancy, or the disease may be followed by more serious digestive disturbances.

Diagnosis.—This is easy only when the whole history can be obtained and the nature of the cause discovered. To distinguish between acute gastric indigestion and *acute gastritis* is usually impossible at the outset and even sometimes later, except at autopsy. Indigestion is usually of shorter duration and less severe. It is also more frequently encountered. Should the severe symptoms persist and vomiting continue after the stomach should have been quite emptied; and, especially if the vomited matter contain blood-streaked mucus, there is reason to believe that gastritis is present. The sudden commencement with vomiting and sometimes convulsions may suggest the onset of pneumonia, influenza, meningitis, or some other *acute febrile disease*; and even later in the attack the presence of the nervous or meningeal symptoms not infrequently attending acute indigestion may make the diagnosis for a time uncertain.

Treatment.—The first indication is to empty the stomach and bowels by abstinence from food and the giving of a purgative. For the latter purpose castor oil is excellent in infancy unless nausea and vomiting be so active that there is reason to fear the drug may not be retained. Calomel in doses of $\frac{1}{10}$ grain (0.006) every half hour until 1 grain (0.07) or less is taken is often of service. No food whatever should be given, but there is no objection to water at the beginning, since, if vomited, it thus produces practically a washing-out of the stomach. Lavage with the stomach-tube is very effective but usually not needed, and the same is true of emetics. If vomiting is troublesome small, repeated doses of bismuth subcarbonate and soda bicarbonate are useful, giving 5 grains (0.32) of the former and 2 (0.13) of the latter at 1 or 2 years of age. A very serviceable remedy consists in teaspoonful doses of equal parts of lime water and cinnamon water given hourly to infants, with larger doses for older children. The application of a hot-water bag or a mustard plaster to the epigastrium is often useful. The return to food should be made cautiously, since relapses are likely to occur. In the case of artificially fed infants albumen water or barley water in small amounts, frequently repeated, is an excellent first food; and, after a considerable interval, the original bottle-mixture diluted with water and lime water and perhaps with the fat omitted. In the case of breast-fed infants the supply of milk should be maintained by systematic pumping, but none given for at least 24 hours and then very little at a time. In older children the first food may be broth free from fat, albumen water, beef-juice, and the like; and later skimmed milk and lime water. The greatest success in the treatment at any age depends upon sufficient thorough starvation and a cautious and slow return to the usual diet. Where there is a tendency to repeated attacks of acute gastric indigestion there is something radically wrong with the diet. In the case of artificially fed infants some modification of the food should be tried. In breast-fed babies the mother's milk should be analyzed, and the effort made to modify this as far as possible, if necessary, by proper dietetic and hygienic measures (see p. 106), or to change the amount given or the frequency of nursing.

ACUTE GASTRITIS

This differs from acute gastric indigestion in the presence of distinct organic alterations. It is of very much less frequent occurrence and, when present, is usually combined with lesions of other parts of the gastro-enteric tract. Many cases supposed to be gastritis exhibit at autopsy none of its lesions.

Etiology.—Diverse causes may be at work, and the disease can be divided accordingly into: (a) the *acute catarrhal*, (b) the *corrosive*, and (c) the *pseudomembranous* forms. The causes in the catarrhal variety are much the same as in acute gastric indigestion, and the disease is more common in infancy than later. In addition to the disturbance of function there is inflammation added, probably dependent upon an infection. In corrosive gastritis the cause is the introduction of an irritant poison as in the case of corrosive esophagitis. Pseudomembranous gastritis, although much oftener seen in children than in adults, is rare. It is usually dependent upon the germ of diphtheria, less frequently on some other germ; and may occur in small-pox, scarlet fever and other infectious diseases, or in sepsis in the new born.

Pathological Anatomy.—(a) In *catarrhal gastritis* the mucous membrane is swollen and reddened and covered with an abundant, thick layer of mucus, often with a brownish stain from slight hemorrhage. The blood-vessels are injected and small punctate hemorrhages are visible. Microscopical examination shows an infiltration of the mucosa with round cells, and sometimes of the submucosa to a limited extent. There may be localized disintegration of the superficial epithelium in scattered patches, or rarely even the production of erosions (*ulcerative gastritis*). Large numbers of bacteria are present in the gastric contents, which consist of mucus and undigested food. The stomach may be either dilated or smaller than normal.

(b) In *corrosive gastritis*, when death has occurred promptly, there are found only the evidences of destruction of the gastric walls, varying in degree according to the intensity of the action, and without signs of inflammation. There may be scattered losses of substance involving the mucous membrane only or extending through the wall of the stomach. If the destructive action has been less intense and life has continued longer, there are found the lesions of acute gastritis combined with scattered, shallow, hemorrhagic erosions of the mucous membrane or deeper ulcerations.

(c) *Pseudomembranous gastritis* exhibits a greyish-green membrane on a part or all of the lining of the stomach, consisting of desquamated epithelium, bacteria, granular matter, and fibrin, and there is an extensive round-celled infiltration of the mucosa and even of the layers beneath. It has been especially studied by Bednar,¹ Orth² and Parrot.³

Symptoms. (a) *Catarrhal Gastritis.*—The onset in infancy is with nausea, vomiting, thirst, gastric pain, high fever, loss of appetite, coated tongue, constipation, somnolence or restlessness, and prostration. The symptoms at first cannot be distinguished from those of acute gastric dyspepsia, but instead of disappearing promptly, efforts at vomiting continue even when no food is taken, the ejected matter containing mucus, sometimes blood-streaked. Abdominal discomfort or pain persists, the tongue remains coated, and the breath is heavy. Fever

¹ Krankh. d. Neugeborenen, 1853, I, 96.

² Lehrbuch d. spec. patholog. Anatomie, 1887, I, 704.

³ Prog. méd., 1875, III, 393.

lessens in degree, but continues present, with great thirst and loss of appetite. Constipation usually gives place to diarrhea, and there is more complete prostration than mere indigestion accounts for. In older children the pain and vomiting are decided features, but the prostration and fever are generally less.

(b) **Corrosive Gastritis.**—The symptoms are very acute and severe. There is the accompanying evidence of inflammation of the mouth, pharynx, and esophagus. Vomiting occurs immediately, the vomited matter being blood-stained. If death does not quickly result, there develop the symptoms of acute catarrhal gastritis in a very severe form, together with those of enteritis.

An analogous condition is seen in the *hemorrhagic erosions (ulcerative gastritis)* sometimes present in septic conditions in the new born, or as an attendant upon severe cases of acute catarrhal gastritis, thrush, or intestinal ulceration. There are no characteristic symptoms distinguishing this from acute gastritis except a greater tendency to gastric hemorrhage.

(c) **Pseudomembranous Gastritis.**—This condition has no characteristics by which it can be distinguished during life. The symptoms may be those of acute catarrhal gastritis or there may be few evidences of disorder of the stomach.

Course and Prognosis.—The symptoms of *catarrhal gastritis* are most severe at the beginning of the attack. Their duration is a few days to a week, but they are liable to be followed by intestinal symptoms. Recovery usually results. Occasionally in infancy an initial convulsion may cause death, or a fatal issue may take place from prostration. Unless properly treated there is great danger, too, of relapse, or of the occurrence of repeated attacks with a final development of a chronic gastritis.

In *corrosive gastritis* the prognosis is very grave. Collapse is liable to result very promptly and death to follow in a few hours after the ingestion of the poison; or life may be prolonged for a time, but death occur in 2 to 3 days from prostration, often with symptoms of a very severe, acute gastritis. Infants nearly always die. Older children may survive and later show evidences of chronic gastritis or of lesions elsewhere in the gastroenteric tract. The prognosis of *pseudomembranous gastritis* is very unfavorable.

Diagnosis.—The recognition of *catarrhal gastritis* is attended by many difficulties. The milder cases cannot be differentiated with certainty from gastric indigestion, and it is only the persistence of symptoms which renders the diagnosis justifiable. The subacute cases strongly suggest typhoid fever, but are to be distinguished in most instances by the more irregular fever, which has a tendency to diminish after the onset instead of to increase. The more severe cases suggest the onset of pneumonia, meningitis, or scarlet fever, but can be recognized by the failure in a short time of any of the characteristic symptoms of these to develop. *Corrosive gastritis* is marked by the extremely sudden and severe onset, collapse, the evidences of corrosion about the mouth, and the history of the ingestion of an irritant substance. The rare cases of erosion from other causes exhibit no distinguishing symptoms except a greater tendency to hemorrhage.

Treatment.—The treatment of *catarrhal gastritis* does not differ from that of acute gastric indigestion. No food should be given at first; small pieces of ice may be swallowed; a free purgative may be administered, if vomiting will permit, in order to empty the stomach and bowels

if the case is seen early or if the giving of food has been persisted with. In some instances lavage is of great benefit, using a normal salt-solution or a 0.5 to 1 per cent. solution of bicarbonate of soda, the latter being selected if the vomited matter continues very acid. Should vomiting be very persistent bismuth may be administered in small doses frequently repeated, or the lime water and cinnamon water combination as in gastric indigestion. The return to food should be even more cautiously attempted than in the milder disease, albumen water or barley water being the first given, or fat-free broth in the case of older children.

The treatment of *corrosive gastritis* consists in the immediate administration of the proper antidote if the case is seen early enough. The stomach should also be thoroughly washed out, but very carefully with due regard to the softened condition of this organ and of the esophagus. After this follows the administration of ice; ice-water; cold demulcent fluids, such as albumen water and solution of gum-arabic; oils; and the like. Prostration is to be overcome by cardiac stimulants hypodermically or whiskey given by the rectum, and pain and repeated vomiting by the hypodermic use of morphine.

CHRONIC GASTRITIS—CHRONIC GASTRIC INDIGESTION

Gastric indigestion frequently repeated or long continued will finally produce distinct lesions. Consequently every case of chronic indigestion may be regarded as based upon an organic as well as a functional disturbance. Certainly no sharp clinical distinction can be made between the functional and the organic disease. It is equally true that there is usually present a combination or even a predominance of intestinal disturbance (*gastroenteritis*). The cases in which this predominance exists are discussed elsewhere (p. 738).

Etiology.—This disease, more common in infants than in older children, is the result of a series of recurrences of acute gastric indigestion, or of a single severe attack of this, or of acute gastritis from which complete recovery had not been made; or it may develop without any previous acute condition. In any event the chronic disorder is often dependent upon persistence with an improper diet. This is especially true in infancy, at which time a food constantly too rich in some ingredient, especially fat, is so often given, or a diet of other substances than milk employed entirely unsuitable for the age. The existence of rickets, tuberculosis, syphilis, or other chronic diseased condition affecting the general nutrition likewise predisposes to the development of chronic gastritis.

In older children the persistent giving of food of an improper character is probably the most frequent cause; but hurried eating with imperfect mastication; eating at irregular times, especially between meals; and imperfect hygiene of any sort, including lack of exercise and sleep, over-fatigue, and undue stimulation of the emotions are very powerful factors. Chronic gastritis may follow also an acute infectious disorder or may attend a chronic debilitating disease. There occurs, too, an inheritance of a predisposition to the malady, and the presence of a neurotic temperament is often an important factor.

Pathological Anatomy.—The stomach is somewhat dilated and the mucous membrane is found covered with a tenacious layer of mucus, exhibits prominent rugæ, is thickened, and is greyish in color; or may exhibit hyperemia in spots combined with punctiform hemorrhages. Small hemorrhagic or other erosions may be seen in severe cases, and occa-

sionally more distinct ulcerations of larger size. Microscopically there is a round-celled infiltration of the mucosa, slight in the milder cases, with compression and partial destruction of the gastric tubules. Numerous bacteria are present in the adherent mucus. The blood-vessels of the mucous and submucous layers are congested, and there may be thickening of the entire wall of the stomach in severe, long-continued cases.

Symptoms.—The symptoms in *infancy* are often somewhat similar to those of acute gastric indigestion, although less severe; but not infrequently the onset is insidious, marked by loss of appetite; coated tongue, occasional nausea and vomiting, and failure of health. Eventually the vomiting becomes the most striking symptom. This may occur promptly after each taking of nourishment, or less often only at longer intervals, the food then being vomited in small amounts and this repeated until the stomach is empty. Often the vomited matter consists only of a very acid, watery liquid, while the solid portion of the food is retained. In long-continued cases in which the condition has advanced to the production of actual inflammation the vomiting of mucus is a prominent feature. This is less marked where the functional disturbance still predominates. The breath has an offensive, very sour odor and eructation of gas of the same character is very common. The tongue is coated and the bowels usually constipated; sometimes diarrheal. The stomach is often greatly distended by gas, resulting in pain and tenderness, restlessness, fretfulness, disturbed sleep, and difficulty in taking food even though the child be evidently hungry. The appetite may be very large but is usually much diminished. Physical examination shows the distended tympanitic gastric region and even sometimes a constant gastric dilatation. (See p. 714.) The employment of the stomach-tube shows the presence of food hours after the organ ought to have been empty. (See Digestion, p. 44.) The contents are in various stages of abnormal decomposition, with mucus and many bacteria. This condition of the gastric contents depends upon the continued disturbance of normal secretion and the inhibition of the gastric motor power, permitting abnormal fermentation; while the inflammation of the mucous membrane produces the abundant mucus. There is nearly always undue acidity, chiefly from the fermentative changes, the hydrochloric acid being generally below normal in amount.

The symptoms described may be nearly continuous, or may vary from time to time; with temporary improvement in which the appetite returns and vomiting is much less, to be followed by recrudescences with evidences of acute gastritis. Meantime the general health gradually deteriorates; there is failure to gain in weight or even loss of it, and anemia is present with the general signs of extreme malnutrition.

In *older children* there is coated tongue, heavy breath, pain or discomfort in the gastric region, and distention and nausea after food. Vomiting, although a common symptom, is not so frequent as in infancy. It may occur after every meal; or only in the early morning before taking food; or irregularly, the vomited matter being the fermented food, always with a large amount of mucus. There is also frequent eructation of gas and perhaps regurgitation of small amounts of the gastric contents between the attacks of vomiting. Appetite may be lost or abnormally great, or there may be a desire for only some articles of diet. The breath is offensive, and constipation is the rule. The so-called "stomach cough" may be one of the chief symptoms. Gradually a certain degree of malnutrition develops, with headache, debility, fretfulness, disturbed

sleep, anemia, emaciation, malaise and a large array of nervous or other indefinite symptoms. In many cases these constitutional manifestations are much more prominent than those giving direct evidence of disorder of the stomach.

Course and Prognosis.—The disease in *infancy* is a severe one, particularly if it has been of long duration. Recovery, however, usually takes place under proper treatment. The earlier this can be instituted the better the prognosis. On the other hand, the younger the infant the less chance it has. The occurrence of the malady in bottle-fed babies and during hot weather adds greatly to the danger. Propitious surroundings make the prognosis more favorable; hospital babies being notable for the liability to contract the disease and for the high death-rate. The course is always tedious and relapses are very prone to occur, without there being any discoverable reason for this in the character of the food given. Finally the disease is dangerous in infancy in the predisposition it creates to the development of diarrheal disorders, rachitis, and infantile atrophy.

In *older children* the prognosis is more favorable so far as life is concerned, although the gastritis is liable to be long-continued, and the tendency to relapse may last for years and perhaps never entirely disappear.

Diagnosis.—This is usually easy, if the history of the case is known. In infants the onset of tuberculous *meningitis* is sometimes marked only by vomiting, lasting, it may be, a number of weeks, and confusion may arise. As a rule the recognition is readily made after a short interval, meningitis showing other characteristic symptoms. *Stenosis of the pylorus* may also readily simulate a chronic gastritis. This is particularly the case when the obstruction has not been absolute, the disease has continued some time, and gastric dilatation has developed. Generally a careful study will distinguish between the two diseases. The active gastric peristalsis and the usual prompt and violent projectile vomiting after taking food characterize stenosis, especially early in the case. The combination of emaciation and cough may suggest *tuberculosis* in older children; but the absence of any localizing evidences of tuberculosis is an indication opposed to the existence of this disease.

Treatment. Infants.—Treatment requires all the thought and skill possible on the part of the physician, since it must vary with the individual case. *Prophylaxis* is much easier than cure, and, on the first warning of danger, as through the repeated development of gastric disturbance, most careful search must be made for the cause of this, which is oftenest a dietetic one; and this removed before the condition becomes established. Overfeeding being of much more common occurrence than underfeeding, it is probable that the cause will be found here; either in too great a total amount of food given, or in one too rich in some particular. In many cases the diet is one entirely unsuited to the infant in question, however fitting it may have proved for others. The effort so often made to feed children by a fixed rule is a fertile source of chronic digestive disorder.

With the *disease already established*, again the search must be for the cause, and this is often a most difficult problem for solution. The whole past dietetic history must be reviewed. This will probably make clear the origin of the disturbance. Perhaps most frequently the fault will be found in an excess of or an intolerance for the fats. In such cases benefit may often be obtained by giving a fat-free mixture, using skimmed milk as a basis for this. In other cases, although much less frequently,

the protein occasions difficulty. In this event, peptonizing may be of service, or the administration of casein-free milk in the form of whey. The latter is, indeed, an invaluable remedy in many instances, but is too weak a food for long continuance. It should be made from skimmed milk when it is desired to avoid fat entirely. Often a diet with high protein-percentage and diminished fat-percentage as obtained is serviceable by the use of some of the numerous "albumin milks," or "casein milks" recommended (p. 148). Buttermilk is a valuable remedy in such instances, being a fat-free food in which the protein is in high percentage, already coagulated and broken up into a fine flocculent state. The addition of a cereal and sugar, as commonly advised (p. 147), adds to its caloric value. In some instances the sugar of milk may give occasion to fermentation and produce the disease. In such its amount must be reduced, or cane-sugar or dextrine-maltose preparations tried as a substitute. Many cases show entire intolerance for milk for a time, and in these the food may temporarily be albumen water or a cereal decoction such as barley water. As this is not sufficient to sustain life indefinitely, a plan often useful is to fortify it after a time by the addition of whey made from skimmed milk, and later by peptonized skimmed milk in small amounts gradually increased. In other cases a cereal decoction may be partially dextrinized (p. 155); and to this milk be finally added in increasing amounts. Malt-soup (p. 156) is often especially useful in such instances. In still other cases the fault is an excess of starchy food, and the chief dietetic treatment consists in the decided reduction of the amount given.

It is usually of benefit in beginning treatment to make a very radical change from the food which had been given. Whatever diet may be selected, the first effort must be to bring about a cessation of the vomiting and other evidences of the disease; but without striving for a gain of weight. Merely to stop the loss is all that is required for a while. After a time, however, the failure to gain properly becomes a matter of importance. The food which has been agreeing is probably much below the normal caloric value required, and a very gradual return to a stronger diet is then imperative. In fact, it is necessary to have constantly in mind the caloric requirements to make gain possible, at the same time not forcing any element of food to an extent which disagrees, since this brings on relapse and retards recovery. The effort should be made to return to a milk-modification containing fat as soon as this can be done with safety.

Apart from the composition of the food its method of administration is important. Some infants do better on small amounts of more concentrated nourishment; others on larger quantities more diluted. As a rule the interval should be long, but only trial will show whether feeding frequently at short intervals and in small amounts may not be better. In some cases food given by gavage will be retained when that taken in the ordinary way is not. In such instances it should usually be in larger amount, often with advantage peptonized, and administered perhaps but 3 times daily. At times the loss of appetite is so great that gavage must be used to sustain life, the infant refusing all food offered to it in the usual manner. In the line of diet there is often nothing so good for young infants with chronic gastritis as the employment of a wet-nurse.

In addition to the correction of diet, lavage of the stomach is a most useful remedial measure. This should be performed once, or sometimes twice, daily; later less frequently, using a normal salt-solution or a 1 per

cent. solution of bicarbonate of soda. Without lavage it is impossible to get rid, to a satisfactory extent, of the tenacious mucus, which otherwise interferes greatly with the access of the digestive secretions to the food taken. Lavage is best performed 2 to 3 hours after a feeding. Sometimes it is of benefit to give food by gavage while the tube is in position for gastric washing; in other cases better results are obtained by giving no nourishment for 2 hours after the washing has been performed. Lavage is, as a rule, well tolerated by infants, and mere feebleness is not a contraindication; but in any infant in whom it produces severe nausea, vomiting, prostration, or cyanosis, it should be employed with great caution or abandoned.

The administration of drugs plays a very secondary part in the treatment of chronic gastritis. Bismuth is sometimes useful in controlling the vomiting, and occasionally it may well be combined with minute doses of calomel, or with benzoate or bicarbonate of soda. In other instances a mixture of soda, bismuth and spearmint water is of value in neutralizing acidity and dislodging accumulated gas. Tincture of nux vomica may be administered with soda in cases where there is great loss of appetite. Occasionally dilute hydrochloric acid with pepsin is of service, particularly where examination of the gastric contents shows diminution of the gastric secretion. Constipation must be overcome by laxatives, especially citrate of magnesia or milk of magnesia. Measures directed to the improvement of the general health must not be forgotten. Among these are the maintenance of the body-temperature, if below normal; the exposure to abundant fresh air out of doors or in a sun-parlor; massage; bodily rest; careful handling after feeding, and the avoidance of all excitement.

Older Children.—Prophylaxis is as important here as in infancy, but difficult on account of the irregularity of symptoms and the often insidious onset. In the treatment of the disease itself, not only is the diet to be carefully regulated, but other matters equally important cared for. Late hours, undue mental excitement or strain, bodily fatigue, too long school-hours, lack of fresh air and exercise, and other possible etiological factors must necessarily be corrected in order to obtain benefit, since so much of the chronic indigestion is of functional origin. Change of climate is often of great benefit. Rest recumbent for an hour daily is of service. No food should be allowed between meals and the diet should be plain and digestible, all highly seasoned dishes, pastry, puddings, cakes and sweet-meats being avoided. No fried food whatever can be allowed, and, as a rule, but a limited amount of carbohydrate given. The diet should consist largely of lean meats; milk not too rich, or preferably koumys or other fermented milk, or buttermilk; small amounts of toast or zwieback; and later green vegetables carefully tried in small amounts. The increase of carbohydrate vegetables should be made slowly and cautiously until recovery is well advanced. Thorough mastication is important. In severe cases a diet purely of milk, modified in some way and often with the fat largely removed, may be required for a time, while in others milk may need to be withdrawn entirely. Tonic remedies are often of benefit, tincture of nux vomica being most useful before food, combined sometimes with soda and with gentian. At times hydrochloric acid after food is better, if the tongue remains heavily coated. If vomiting is troublesome bismuth or the combination of liquor calcis and aqua cinnamomi, previously referred to (p. 720) may be of service. Chronic constipation must be relieved by appropriate remedies. Sometimes the

addition of small amounts of an aromatic, such as tincture of ginger, relieves pain by causing displacement of accumulated gas. Constant care for the general hygiene and the diet is required often for years, in order to avoid the great tendency to relapse.

CHAPTER V

DISEASES OF THE STOMACH AND INTESTINES (CONTINUED)

TYMPANITES

Distention of the intestine and stomach with gas is a symptom which may attend various diverse conditions, and in many instances becomes the most important one demanding special treatment. It may depend directly upon digestive disturbance, being one of the most prominent symptoms of chronic intestinal indigestion, and due to the fermentation of food especially of an amylaceous nature. It is a very constant and pronounced symptom in acute peritonitis and sometimes in appendicitis, the result in each case of a temporary paralysis of the intestinal wall. Tuberculous peritonitis is likewise attended by it. Typhoid fever exhibits it at times, especially in older children, but seldom to the extent seen in adult life. In pneumonia it is sometimes excessive and constitutes a serious symptom with a grave prognostic import. Rachitis constantly is attended by tympanitic distention of the abdomen, dependent partly upon the accompanying intestinal catarrh and partly upon the weakened abdominal and intestinal walls. Finally congenital dilatation of the colon is characterized by a remarkable dilatation of the intestine by gas.

The **treatment** consists on the one hand of the cause, and on the other is symptomatic, intended to relieve the distress often present. In the latter category are such measures as turpentine stupes externally; intestinal douching; the use of the rectal tube, which may be allowed to remain in position even for some hours if needed; the administration of carminatives and of asafetida; and in very urgent cases the hypodermic administration of eserine. I have obtained success with this last when other measures had failed in very threatening conditions. As it is, however, a remedy which can exercise a powerful depressing influence it may well be combined with strychnine.

Opium, although a reliever of pain, should be given cautiously when tympanites is present, since it is likely to increase the distention by diminishing intestinal peristalsis.

INTESTINAL COLIC

(Enteralgia)

Although but a symptom, colic is one so important and frequent, especially in infancy, that it deserves separate consideration. In the narrower sense it consists in the occurrence of intestinal pain in *paroxysms*, depending sometimes on distention, oftener upon a spasmodic contraction of the muscular wall of the intestine. This paroxysmal nature distinguishes it from the more persistent pain which may accompany any inflammatory condition of the intestine or peritoneum, or some nervous disorder, and which is to be included under the broader title of *enteralgia*.

Etiology.—Among the various causes the most common is any form of intestinal indigestion with the resulting production of gas. This is observed with especial frequency in the first 3 or 4 months of life, and occurs in breast-fed babies as well as others, even when analysis of the breast-milk shows nothing abnormal. The addition of starch to the diet is a fertile source of pain in many bottle-fed children, although any of the elements of the milk itself may produce it. In older children colic may attend acute intestinal indigestion from the eating of green fruits and other unsuitable substances.

Many cases of colic appear to have a nervous origin, brought about reflexly as, for example, through chilling of the surface of the body. The ingestion of certain poisonous substances, such as lead or arsenic, may produce intestinal pain. This is very uncommon in infancy, but an analogous condition occasionally follows from the nursing of breast-milk which has been secreted under disturbed psychic influences. Purgative drugs are also a frequent source of pain in the intestine. Peritonitis, enteritis of any form, appendicitis, intussusception, and any condition which produces tympanites may be productive of enteralgia.

Symptoms.—In enteralgia in general the chief symptom is abdominal pain which arises in the intestine. In true colic this is paroxysmal. The infant may exhibit other symptoms of indigestion; but very frequently, especially in breast-fed babies, it is healthy and thriving except for the colic. The attack begins more or less suddenly, perhaps after a short period of discomfort. The cry is very loud and unceasing; the face is congested and often somewhat cyanotic, or with pallor about the mouth; the abdomen is distended and tense; the legs are now drawn up upon the abdomen, now momentarily extended; the feet are often cold; the hands are clenched and the arms flexed and drawn to the body. The paroxysm continues a variable time, sometimes several hours with complete or partial intermissions lasting for a few moments only. Finally with the expulsion of gas or feces the symptoms disappear completely and the infant falls asleep. If the colic has been prolonged and intense quite a degree of prostration may follow. In many instances the symptoms are not nearly so severe and the baby is merely fretful and wakeful until relieved. In others, with highly sensitive nervous systems, convulsions may develop.

The frequency of the occurrence of colic varies greatly. In many it is only occasional, but in others in the first few months of life it seems oftener present than absent, and is especially liable to occur in the night-time; with the result that the parents, as well as the infant, obtain almost no sleep. It is a noteworthy fact that whereas every one in attendance seems exhausted on the next day, the infant often appears none the worse for its experience.

Diagnosis.—This is easy in most cases if the attack is seen by the physician. Oftener, however, it is difficult if dependence must be placed entirely on a description given by the mother or nurse. Colic is especially to be distinguished from hunger. The cry of the former is generally sharper, more violent, and more paroxysmal; that of the latter more persistent and often more fretful. Frequently the infant with colic refuses food; in other cases it will take it well if the pain is not too severe, and may be temporarily relieved by it. Soon, however, the cry returns in full force, thus excluding completely the diagnosis of hunger. Earache causes very persistent screaming, and there is tenderness about the ear. The pain of peritonitis and appendicitis is to be distinguished from colic

by the more persistent character and by other attendant symptoms, especially the tenderness on pressure. In colic gentle pressure is often a source of relief. The pain of intestinal colic is often difficult to differentiate from that of gastralgia, which, indeed, it may attend, or with which it may alternate. Older children refer the pain of gastralgia to the epigastrium. In infancy relief of pain by expulsion of gas from the rectum indicates that the disturbance was in the large intestine, which is the most common situation.

Treatment.—The occurrence of the disease must be prevented, as far as possible, by a careful study of the diet, and a change in this if it is found necessary. Whether advisable or not depends upon the frequency and severity of the paroxysms and upon the general health of the infant in other respects. If an infant is thriving upon its mother's milk, except for frequent attacks of pain, and the milk is found on analysis to be practically of normal composition, it is usually better to temporize, and to give what relief is possible when pain is present, merely altering, perhaps, the intervals and length of nursings; especially since the tendency is for colic to lessen greatly after the first 4 months of life. In such infants must be considered the possibility of the pain being largely neuralgic rather than due to indigestion; and, in any event, weaning threatens more serious dangers than the colic presents. Should, however, the infant be manifestly losing health, weaning is to be recommended; and in bottle-fed babies with severe colic some change in the diet is certainly advisable. The prevention of attacks is also to be attained by avoiding chilling of the surface, by seeing that the bowels are opened regularly, and especially that a movement is obtained shortly before the time of day when the pain comes on or is worst. An enema may be used for this purpose. Giving a carminative such as sodamint or some modification of it before the meals is also often a useful preventive measure.

During the paroxysm a hot application should be placed on the abdomen, such as a hot water bag, weak mustard plaster, turpentine stupe, or spice plaster. This tends to relax the spasmodic contraction of the intestinal muscles. Rubbing the abdomen with the warm hand is often of service. One of the best remedies is the giving of an enema, since this starts the peristalsis and causes an expulsion of feces and of gas. The injection may consist of soap and water, using 6 or 8 ounces (177 or 237) or more according to the age of the child (p. 234). Internally carminatives are useful, not only because they are effectual in producing eructation of the gas, should the condition be one of gastric distention and spasm rather than intestinal colic, but because in the latter condition they act reflexly from the stomach, increasing the intestinal peristalsis and causing the gas to be expelled from the bowel. Bicarbonate of soda may be given with spearmint-water, peppermint-water, or fennel-water. In more severe cases in young infants the addition of the bromides (1 to 2 gr.) (0.07 to 0.13), or of chloral ($\frac{1}{4}$ gr.) (0.016) is very effective in relaxing the muscular spasm. Mistura asafetida (10 m.) (0.62) or *spir. aether. comp.* (4 to 5 m.) (0.25 to 0.31) is often very serviceable. In the worst cases opiates should be used; yet bearing in mind that if the colic is due to flatulent dyspepsia rather than to a purely nervous condition, relief follows but peristalsis is inhibited, constipation occurs, and flatulence may be finally increased.

THE FECES IN DIGESTIVE DISEASES

In older children the character of the abnormal stools varies with the food taken; meat-fibre, vegetable material, milk, and the like showing themselves in different degrees. Mucus, too, may be in large amount, especially in some forms of chronic intestinal indigestion and in colitis. The following description applies especially to the stools of infants:

The normal stool of the breast-fed infant is mustard-yellow in color, smooth, with no evidences of undigested food, and slightly acid. (See p. 46, Fig. 12.) In healthy artificially fed children the shade of color is often somewhat lighter, depending upon the amount of fat in the food; a high percentage of this producing a paler stool. At the most there may be small, scattered white masses of undigested fat. In older infants the yellow is somewhat deeper and the stools more salve-like in consistency.

Mucous Stools.—In cases where food has been withdrawn for a day or two, the stools consist of the thin mucoid secretion of the intestine stained a brownish tint (*hunger stools*). After a purgative, especially castor oil, a large amount of mucus is passed in infancy, wrongly supposed by the mother to have been present before the oil was administered. Mucus occurs readily, too, in many disturbances of the digestive tract in early life, and may, at first, if continuing but a short time, indicate either an inflammatory or a functional disturbance of the large intestine. If it is persistent, it points rather to inflammation. Undigested starch has a certain similarity in appearance to mucus, but can be distinguished from it by the iodine reaction. Stools composed almost entirely of blood-stained mucus occur in dysenteric conditions and in intussusception.

Protein Stools.—These are seen in infants especially where the protein of the food is of high percentage and undigested. The odor of putrefaction is discoverable at times, combined with an alkaline reaction. The color is brownish-yellow, and mucus is always present. Sometimes tough, yellowish protein-curds are found (Fig. 243).

Fatty Stools.—Fat may show itself either as soap; or in the form of a smooth, yellow stool; or as soft white curds composed of neutral fat.

The *soap stool* depends upon a large excess of fatty acids, combined with calcium or magnesium to form a soap. They are white or grey, shiny, fairly firm, homogeneous, crumbly or salve-like, of acid reaction, and have a rancid or sour odor (Fig. 244). They are commonly combined with more or less protein, and, if this is in large amount, the odor is cheesy or offensive from the decomposition of this, and the reaction may be alkaline.

The *fatty stool* is of a bright-yellow color, soft, and of greasy appearance, and will produce a grease spot if placed upon paper. It contains a large amount of neutral fat and fatty acids. The stools are thin, and may be frequent enough to produce a fatty diarrhea.

The *curdy stool* exhibits numerous large or small curds, and is of an acid reaction. The curds are generally soft, white, and composed of fat. They are to be distinguished from the yellow curds consisting of proteid material and already referred to. The stool as a whole, apart from the white lumps, is of a green or yellowish color and often diarrheal, and mucus is always present (Fig. 245). The presence of curdy stools, as also of soap stools, is a matter of little clinical consequence unless symptoms of indigestion are present (Talbot).¹

¹ Bost. Med. and Surg. Jour., 1918, CLXXIX, 35.

Carbohydrate Stools.—Very often the stool of this nature is of a normal consistence, homogeneous, smooth, and of a brown or yellowish-brown tint and acid reaction (Fig. 246). If starch has been administered, this may perhaps be found with the iodine test. In other cases there may be a decomposition of the carbohydrates in the intestine, producing thin, frothy, acid stools, often green in color. The odor is then sometimes that of acetic acid.

Green Stools.—These are of very common occurrence. The stool may be of a faint pea-green color when passed, or may become so shortly afterward. This probably depends upon unaltered biliverdin, and evacuations of this nature are not to be considered pathological. In other cases the color is of a deep spinach-green, seen chiefly in the mucus passed in the stool (Fig. 247). In some cases this probably depends upon the action of a specific microörganism. These green stools very frequently have present the white curdy masses consisting of fat already described. Green, watery stools are often seen in acute intestinal indigestion, both in breast-fed and artificially fed infants. They may depend either upon an excess of fat or of sugar.

Brownish Stools.—As stated, these are quite characteristic of many cases where food containing a high percentage of protein or of carbohydrate (Fig. 246) is given. Children fed on whey develop brownish stools, and the hunger-stools referred to (p. 731) have a similar tint.

Blood in the Stools.—This is not necessarily a serious matter. Any moderate congestion of the mucous membrane of the large intestine may develop streaks of blood upon the mucus passed (Fig. 247). Blood streaks may depend, too, upon hemorrhoids or fissure of the anus, or upon the passage of a large constipated movement. Combined with a considerable amount of mucus, blood is also seen in intussusception and in ileocolitis. If in large amount and coming from higher in the alimentary canal, the stools are colored a reddish-black, and may be the result of ulceration or of hemorrhage from other causes. The condition is to be distinguished from the black stools dependent upon the administration of bismuth or iron.

Intestinal Sand.—Occasionally minute sand-like bodies are found in the stools, and may be in considerable quantity. They may be visible when the passages are of a diarrheal nature, or discoverable only after washing and straining them from the fecal matter. The nature of the sand would seem to vary. In some instances it has appeared to consist of the woody cells from the banana; in others it is of a crystalline nature, probably produced in the process of digestion. It is uncertain whether any symptoms are attendant upon the presence of intestinal sand.

Micro-chemical Examination of the Stools.—This procedure is an aid in determining the nature of the stools. Starch is detected by the application to the feces on a glass-slip of a little diluted Lugol's solution, which colors the granules blue. The test for the comparative amount of fat and its nature is the most important. The following description is based chiefly upon the contributions of Talbot.¹ A minute portion of the stool is placed upon a glass-slip, stained with a saturated 95 per cent. alcoholic solution of Sudan III and covered with a cover-glass. Another portion is similarly stained with a saturated solution of carbolfuchsin,

¹ Arch. of Ped., 1911, XXVIII, 120. Amer. Journ. Dis. Child., 1911, I, 173.

diluted one-half if necessary. The following table shows the results obtained:

Stain	Neutral fat	Fatty acids	Soaps
Sudan III.	Drops staining red.	Drops staining red, or crystals which may or may not stain.	Do not stain.
Carbolfuchsin. . . .	Do not stain. Remain oily, colorless drops.	Stain brilliant red.	Stain dull red.

After this examination is over, a drop of glacial acetic acid is allowed to run under the cover-glass of the Sudan III slide, and gentle heat is applied until bubbling begins. This turns the neutral fat and soap into fatty acids in the form of large red drops while hot, crystals when cold; and an idea can be obtained of the total amount of fat present. Whether or not this is normal may be estimated as follows:

Entire digestion of fat, enlargement about 400 diameters, Sudan III, followed by acid and heat gives only 1 to 3 fat-drops in the field; *normal digestion of fat*, under the same conditions, gives 5 to 8 drops; *slight excess of fat*, gives 8 to 10 drops; *moderate excess of fat*, gives over 12 drops; *Large excess of fat*, practically the whole slide is filled with fat-drops.

Talbot,¹ however, is of the opinion that the micro-chemical examinations of the stools is usually of little importance unless attended by manifest symptoms.

DIARRHEAL DISORDERS

Like vomiting diarrhea is only a symptom, but one of such importance that a review of its various causes and characteristics is necessary. It is one of the most frequent and often most serious of the disorders of childhood and especially of infancy. The part which it plays in the general mortality of early life has already been referred to to some extent (pp. 213-216). Hermann² estimated that more than 33 per cent. of the deaths in the 1st year of life occurring in Berlin were the result of digestive diseases, chiefly diarrheal. According to the statistics given by Still,³ from 2000 to 4000 infants under 1 year of age died annually in London from diarrheal diseases, or 18.88 per cent. of 90,823 total deaths in the 1st year of life during a period of 5 years observation. The deaths from diarrheal disease as compared with other affections of children is well shown in the diagram given under the Causes of Death (p. 216, Fig. 31).

Etiology.—*Age* is consequently a predisposing factor of importance, the great majority of cases occurring in the first 2 years of life, and the tendency to the disease diminishing greatly after this period. Season, too, is a very important matter, the summer being the time of year in which the greater number of cases occur. The incidence and the mortality, indeed, seem often directly proportionate to the heat of the weather (Fig. 248). (See also p. 213, Fig. 30.) Poverty, crowding, uncleanness, and previously debilitated health, such as prevail so extensively among the poor children of cities, are hygienic factors of great importance. The influence

¹ Bost. Med. and Surg. Journ., 1918, CLXXIX, 35.

² Zeitschr. f. Socialwissenschaft, VII, 4, 238. Ref., Ebert, Jahrb. f. Kinderh., 1905, LXI, 500.

³ Common Disorders and Diseases of Childhood, 1909, 210.

of the *die* in the 1st year of life is of especial significance. As already stated (see Mortality, p. 212), the liability to death is far greater among the artificially fed infants, and this is particularly true when the food is improperly prepared or contains bacteria. Other diseases frequently have diarrhea as one of the chief or secondary symptoms. This is true of the acute infectious diseases, especially typhoid fever and of measles, but sometimes also of scarlet fever, diphtheria, poliomyelitis, pneumonia, septicemia, and others. In addition to the factors mentioned, other causes of various sorts may be operative, and a classification might

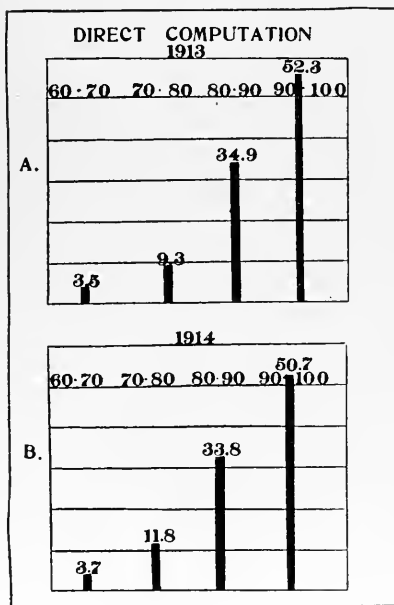


FIG. 248.—DIAGRAM SHOWING THE RELATION OF HEAT TO THE ONSET OF DIARRHEA AMONG 86 (1913) AND 136 (1914) DISPENSARY INFANTS IN ST. LOUIS.

The figures above the vertical columns give the percentage of the total cases which developed with the temperature of the upper line. (*Bleyer, Jour. Amer. Med. Assoc.*, 1915, LXV, 2161.)

is associated with gastric disturbance of a similar origin and nature, the causative influence being something ingested which disorders the stomach first and then passes into the bowel; or both regions being affected at the same time and each to a degree which varies with the individual case.

The following classification may be employed as a matter of convenience:

1. Diarrhea Due to Locally Acting Mechanical or Chemical Causes.—

These two factors are so closely allied in their action that they may be considered together. Purely *mechanical* causes are those which induce increased secretion and peristalsis through their mechanical irritation. The presence of a large mass of undigested food in the intestine may act in this way. This is particularly true if the diet has consisted of such substances as unripe fruits; green corn; tough vegetable matter, such as

be made accordingly; but inasmuch as often several causes are combined in one case, the relationships are close, and no very sharp division can be drawn. From a pathological standpoint, also, the varieties of diarrhea are intimately related, there being in all a functional disturbance, and in the severer forms an organic change as well; or the latter developing in the later stages of a diarrhea which was at first only functional in nature. Further, in all periods of infancy and childhood diarrhea is accompanied by an increase of the number of bacteria in the stools; and it is consequently difficult to determine whether the condition is then the result of a directly infectious influence of the germs upon the mucous membrane, or of their action in causing decomposition in the food, with consequent production of toxic substances; or whether the increase of germs is secondary to a simple disturbance of function of which the diarrhea is a symptom. In fact from every standpoint it is often quite impossible to distinguish with certainty the different forms of diarrheal affections, or to understand their mode of production. Quite commonly, too, diarrhea

asparagus stalks and celery; food containing a large amount of not easily digested matter, such as cabbage, turnips, grape-seeds and grape-skins, fig-seeds and the like. Certain of these act also as *chemical* causes of diarrhea. This is probably true, for instance, of unripe fruit, cabbage, plums and similar substances. In infants some of the food elements may be directly injurious to the digestive functions. As the result of chemical causes is to be classed, also, the diarrhea depending upon the action of medicines, as when too large a dose of some purgative sets up an irritation which lasts beyond the ordinary influence of the drug.

2. Diarrhea of Toxic Origin (Acute Gastroenteric Intoxication).—This depends upon the direct influence upon the intestine of the toxic substances produced by the agency of bacteria. These toxins, it is true, are also absorbed and occasion constitutional symptoms and even organic changes in the organs; yet their action, so far as diarrhea is concerned, is a local one and is, strictly speaking, of a chemical nature, producing functional disturbances together with a degree of catarrhal inflammation.

In theory the difference between this and the preceding class of cases is that bacterial action predominates in the second group. In the first class the food taken may be irritating and act directly upon the intestine; in the other the bacteria produce chemical changes in the food which are irritating to the mucous membrane. The distinction, therefore, is not a sharply defined one.

3. Diarrhea of Nervous Origin.—This is a common form at all ages of life. To be placed here are, for example, the cases due to such causes as chilling of the body, the action of very hot weather upon the organism, emotional excitement, fatigue, and the reflex action often immediately following the taking of food into the stomach.

4. Diarrhea of Acute Intestinal Indigestion.—This is a form of very great frequency. It belongs partly to one or the other of the classes just described, and like them is dependent chiefly upon a functional disturbance of the intestine.

5. Diarrhea of Metabolic Origin.—Omitting from this category cases the result of the local toxic action of substances upon the intestine, diarrheas of this class are in reality eliminative, produced by the efforts of the system to get rid of some poison in the circulation. Most of the diarrheas seen in the acute infectious diseases are of this sort. Uremia produces diarrhea in the same way.

6. Diarrhea of Inflammatory Nature.—Here the condition is the direct result of organic intestinal changes and the irritation and disturbance of function which these occasion. In this class are diarrheas dependent upon ileocolitis. This disease frequently belongs primarily to the group of toxic diarrheas, the disturbance being largely functional; but later decided inflammatory changes develop. The results of the ulcerative lesions of tuberculous enteritis are to be placed also in the category of inflammatory diarrhea.

Symptoms and Treatment.—Acute intestinal indigestion, acute gastroenteric intoxication and ileocolitis are diseases of such importance in early life that they must receive separate consideration. Other forms of diarrhea exhibit symptoms and require treatment of a nature in accordance with the cause. That from local mechanical or chemical causes is attended by gastric disturbance, abdominal pain, and often fever. Unloading of the bowel by a freely acting purgative is required, as is the temporary withdrawal of food. When there has been decided irritation or much pain, opiates may be needed later. Diarrhea of nerv-

ous origin has few symptoms except the looseness of the bowels. It demands the removal of the cause in order to prevent recurrence of the attacks, and for the attack itself opium may be given promptly. On the other hand, in diarrheas of an eliminative nature no opium should be administered early unless the condition is so severe that exhaustion is feared. Nature's efforts at elimination must not be interfered with. Cases of diarrhea of any sort should be treated by rest in bed if at all severe, as exercise tends to prolong the attack. Inasmuch as there is a large loss of the salts of the body in severe diarrhea, hypodermoclysis with normal salt solution is often of great value in supplying the needed sodium chloride and favoring the retention of liquid in the system.

ACUTE INTESTINAL INDIGESTION

Under acute intestinal indigestion may be included those milder cases of digestive disorder dependent upon functional disturbance produced in various ways, in which the local symptoms generally predominate, usually with little if any evidence of any toxic involvement of the organism. The distinction between this and the gastroenteric intoxication to be next described is one chiefly of degree, and there are intermediate forms which unite the two so closely that a division can be made only for the sake of convenience of study. Acute intestinal indigestion is also quite commonly associated, especially in infancy, with the acute gastric indigestion already considered. It corresponds in many respects to the Dyspepsia of Finkelstein's classification (p. 698).

Etiology.—Among the principal causes in *infancy* are overloading of the gastrointestinal canal with too large an amount of food; the use of a diet unsuited to the patient, such as an excess of protein, fat, or sugar; nervous or other conditions affecting the milk of the mother; and influences involving the digestive power of the infant, such as undue excitement, acute diseases, rachitis and the like. The effect of hot summer weather is especially noteworthy and particularly so in artificially fed infants, not only through the alteration of the intestinal contents produced by bacterial growth, but through the direct prostrating effect of the high air-temperature upon the child's digestive powers. In *older children* are seen such causes as the ingestion of unripe fruits or indigestible vegetables; fatigue; acute illnesses; chilling of the skin; very hot weather; and similar causes temporarily inhibiting the digestive functions. Sometimes one article of food always produces diarrhea in a certain child, although harmless to others. This may be true, for instance, of fish, shellfish, certain vegetables or fruits, and even milk. Bacteria would appear to play a very minor rôle, so far as any direct effect upon the intestinal mucous membrane is concerned; although doubtless their action in altering the character of the food taken is of importance.

Pathological Anatomy.—There are no lesions other than congestion of the mucous membrane; but the action of the etiological agent, whether directly or through reflex disturbance, produces an increase of the intestinal secretion with outpouring of liquid from the vessels and an augmented peristalsis, all resulting in diarrhea.

Symptoms.—The disease is usually associated with gastric symptoms, especially in infancy, the noxious agent, if of the nature of an indigestible article, exercising its action upon the stomach before it reaches the intestine. Consequently in this event the attack is usually ushered in by vomiting of short duration; and this is attended or soon followed by fever, even up to 104° or 105°F. (40° or 40.6°C.) but disappearing generally

within 24 hours; sometimes persisting a longer time (Fig. 249). There are intestinal pain (see Colic, p. 728); often distention of the abdomen by gas; restlessness; slight prostration, and soon diarrhea. In older children the symptoms of the so-called "bilious dyspepsia" are present, consisting of loss of appetite; nausea; vomiting; diarrhea; headache; occasionally slight jaundice, and moderate temporary fever, if any. The amount of urine is much diminished if the diarrhea is severe.

The stools in infancy contain undigested milk in larger or smaller curdy masses in a watery fluid, sometimes with more or less mucus, but without blood. The color varies from yellow to white or green, the latter depending upon unchanged biliary coloring matter (biliverdin). The stools are especially liable to be passed after taking food, and are usually less frequent at night. The number is always increased, from a few larger up to 15 or 20 quite small stools in 24 hours; the odor may be sour or fetid; the consistence is liquid, or sometimes frothy; the reaction is generally acid. Only bacteria native to the intestinal canal are present, although these may be increased in number.

In older children the stools often contain at first undigested food; and sometimes the particular article of diet which has caused the attack is easily recognized. Later they are chiefly watery, of a yellow or brownish color, and with an offensive odor.

Course and Prognosis.—Under proper treatment the course is generally short, and the attack is over in a few days. It is usually, only when the disease has attacked infants during hot weather that there is danger of the condition passing into one of gastroenteric intoxication or ileocolitis. There is frequently, however, a tendency for repeated attacks to occur. Sometimes, too, susceptible infants die from convulsions, but as a rule the prognosis is good.

Diagnosis.—This rests especially upon the diarrhea, the shortness of the course, and the absence of toxic symptoms. Early in the attack the disease cannot with certainty be distinguished from acute gastroenteric intoxication, which is, indeed, so frequent a sequel unless great care is taken.

Treatment.—The primary indications are to stop the supply of food and to empty the bowels. The first is accomplished in infancy by administering thin barley water or even simply water for 24 hours or more, and then employing albumen-water or broths, or a stronger barley de-

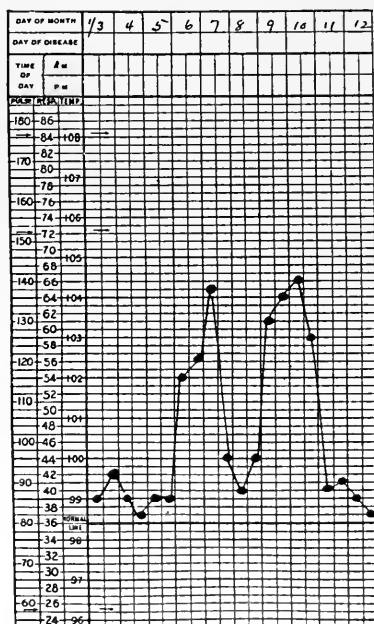


FIG. 249.—ACUTE GASTRO-ENTERIC INDIGESTION FOLLOWING CHANGE OF FOOD.

Leonard F., aged 10 months. Suffered from vomiting for some weeks. Admitted to Children's Ward of the Hospital of the University of Pennsylvania on Jan. 2, suffering from rickets; Jan. 6, has been doing well, but ceased to gain, change of diet on this date caused vomiting, liquid greenish movements and fever; Jan. 11, fever ceased; condition of stools had improved.

coction for 3 or 4 days more. It is of the greatest importance that the return to milk be made very gradually, using at first diluted skimmed milk or a weak modification of whole milk, and then carefully increasing the strength of this.

Although the bowel may be emptied by an enema, and should be so treated if the case is seen early and there is much abdominal distention, this is not sufficient, and purgation is required in order to empty the small intestine as well as the colon. Castor oil or calomel are the best drugs if the stomach is tolerant. The dose of calomel is practically the same for any period of life; namely $\frac{1}{8}$ grain (0.008) every $\frac{1}{2}$ to 1 hour until 10 doses are taken, followed by some saline. For this purpose, or used without the calomel, milk of magnesia, or citrate of magnesia for older children, is very efficacious. If diarrhea persists astringents must be employed, especially bismuth, perhaps with small doses of opium if required; but this treatment must not be used as long as fever, abdominal distention, and nervous symptoms of any nature are present, since these are often an indication that the bowel is not yet emptied of the harmful contents and that the disturbance of the digestion is still maintained, or that more serious inflammation or intoxication is occurring. Stimulants are seldom required, and only if there is much prostration.

ACUTE GASTROENTERIC INTOXICATION AND INFECTION

(Summer diarrhea; Milk poisoning, Infective diarrhea; Acute Gastroenteritis; Cholera Infantum; Food-intoxication)

This most serious and very common affection of early life, especially of infancy, has been described under various names. As already pointed out, there are no sharp etiological or pathological lines of distinction separating it from the acute intestinal indigestion described (p. 736); the latter being only a milder form of the former, and the two disorders shading into each other. Clinically the distinction rests upon the greater severity of the local symptoms and the constitutional involvement. It is still a matter of dispute to what extent the disease is chemical or bacteriological respectively in its origin.

Etiology.—The *predisposing causes* are various. The previous presence of acute intestinal indigestion is prominent here, and the influences which lead to this. Among them are defective hygiene, debilitated health, and especially the character of the food. The disease is far more common in infants fed artificially than in those at the breast. (See Mortality, p. 212.) The excessively hot weather of summer time is a very important factor, its action being partly through the depressing effect upon the infant and the consequent interference with the digestive functions, partly through the favoring of the rapid growth of bacteria in the food (Fig. 248). The number of cases of diarrhea is often, indeed, in direct proportion to the existence of extremely warm weather. Age, too, predisposes, the disorder being far more common in infancy. There is, however, no inherent special tendency to it in the "second summer," other than the fact that more infants are artificially fed at that period. Undoubtedly, too, conditions affecting the general health, such as acute diseases, rachitis, and even a constitutional debility predispose.

The *exciting cause* of the disease would appear to be chiefly the influence of toxins. It is probable that in some instances these toxins are produced by bacterial action upon the milk before it is ingested. In accord

with this view is the occurrence of the disorder after the ingestion of impure milk which has been sterilized; the bacteria having been killed but the toxins remaining. In other cases the bacteria normally present in the intestinal canal (p. 45) probably take on increased multiplication and virulence, and are the cause of increased production of toxins which act both locally and by absorption. In others germs foreign to the normal intestinal flora are found in large numbers, and in still others it is probable that the continued employment of unsuitable food operates chemically without the necessity of abnormal bacterial action taking place.

The microorganisms which have been oftenest accused are the streptococcus enteriditis (Booker),¹ the bacillus coli (Escherich),² the bacillus pyocyaneus, bacillus enteriditis, bacillus dysenteriae, bacillus aërogenes capsulatus, and the bacillus proteus vulgaris. It is quite certain that no one germ is specific, and just what etiological relationship the bacteria hold to the disease is still uncertain. The subject is rendered more complicated by the fact that even including ileocolitis, which appears to be a definite bacterial infection, there is no one complex of symptoms which alone is brought about by a single species of microorganism, and, conversely, one species is clearly capable of producing different clinical manifestations.

Pathological Anatomy.—The changes are chiefly degenerative, in contradistinction to ileocolitis where inflammatory lesions predominate. They may affect the whole gastrointestinal tract. The stomach is usually distended with gas. Its mucous membrane is slightly thickened, injected, and of a bright-red color in patches, or it may be anemic. Parts of the rugae project more than normal and these are the regions oftenest reddened. Minute hemorrhages are sometimes visible, and abundant secretion of mucus may cover the gastric walls. A similar condition obtains in both the small and the large intestine, the ileum being the portion oftenest most involved, showing marked congestion in some portions while other areas may be unusually pale. The solitary follicles and Peyer's patches are abnormally prominent and generally congested. It is noteworthy, however, how slight the macroscopic lesions may often be, even when the symptoms have been severe.

Microscopically there is found a degeneration of the epithelial cells of the mucous membrane, varying in degree with the intensity of the toxic action. This is the characteristic lesion of the disease. In addition severer cases exhibit more or less desquamation producing loss of the epithelium in many places. There is, further, a small-celled infiltration of the mucous membrane including Peyer's patches and the solitary follicles. Bacteria penetrate the intestinal wall only in the regions where there has been loss of the epithelial lining (Booker).³ They may even reach the lymphatic glands and channels, the peritoneum, or other parts of the body, and even occasionally give rise to symptoms of septicemia. Some of the organs may at times show the result of the action of the toxins. Thus the liver may exhibit fatty degeneration of its cells; the kidneys some degree of cellular degeneration in severe cases; the mesenteric glands are often enlarged; bronchopneumonia is not infrequent and occasionally cerebral changes may occur. The pathological condition

¹ Johns Hopkins Hosp. Rep., 1893, VI.

² Escherich and Pfandler, in Kolle and Wassermann Handb. d. pathog. Microorg., 1902, 433; also 17 Congr. f. inner. Med., 1899, 125. Ref., Czerny and Keller, 192.

³ Loc. cit.

shades gradually into the characteristics of ileocolitis; the inflammatory infiltration and ulceration being greater in the latter and the colon being the region principally affected.

Classification.—The disease may affect either the stomach or the intestines, or many diverse symptoms connected with other parts of the body may assume especial importance; but in general, cases may be divided into three classes not sharply separated: (1) The ordinary type; (2) acute milk-poisoning; (3) choleric form diarrhea; it being understood, however, that these are only somewhat different clinical manifestations of a single disorder.

1. THE ORDINARY TYPE.—This may develop either in infants or in older children. The symptoms vary according as the stomach or the intestine bears the brunt of the attack. In *infancy* the stomach is generally involved to some extent at least, the symptoms manifesting themselves gradually after some evidences of indigestion; or quite suddenly with fever of 101° to 103° F. (38.3° C. to 39.4° C.) or sometimes 105° to 106° F. (40.6° to 41.1° C.), combined with colic and vomiting. The nervous symptoms are early in appearing. They may consist only of restlessness, irritability, and fever; but not infrequently there is great prostration, sunken eyes, unconsciousness, or convulsions, and the child appears very ill. There is loss of appetite; but sometimes great thirst causes liquid nourishment from a bottle to be taken readily. The vomiting may be moderate and soon cease or be even entirely absent, or it may be severe and very persistent. The vomited matter is at first the food taken, and later even any water swallowed, or may consist of mucus or bile. Generally vomiting stops soon or becomes a minor symptom. Diarrhea develops, as a rule, within 24 hours. The movements are at first chiefly fecal, often with white curdy masses; but later may become very liquid, of a greenish or pale-yellow color and of an offensive odor and contain but small amounts of fecal matter. A large quantity of gas is often passed by the rectum. The stools vary from 2 to 3 to 15 or 20 in 24 hours. They are very often preceded by pain and expelled with force and sometimes with moderate straining, and with more or less mucus; but this condition is not as marked as in ileocolitis. The development of diarrhea is often attended by a diminution or subsidence of fever and an improvement in the nervous symptoms; but if the case continues and if the stools are large, wasting of the body rapidly takes place, the pulse is accelerated and weak, and the prostration is unchanged or grows worse. The urine is usually scanty and often contains albumin, and in severe cases casts.

In *older children* the onset is oftenest abrupt, vomiting may occur but is less frequent than in infancy, and abdominal pain is a more prominent symptom. The nervous symptoms likewise are usually less marked and the temperature is not so high. The stools are very offensive, liquid, and usually of a brownish color.

Course and Prognosis of the Ordinary Type.—In the more favorable cases, properly treated, the severity of the general symptoms is lessened and the temperature falls when diarrhea begins, and after this has lasted a week or more the stools gradually grow less frequent and of a better character and convalescence is established. Not infrequently, however, the fever lessens but does not disappear, and runs an irregular course, the abnormal stools persist, and the attack assumes a subacute form, with difficulty distinguishable from the later stages of ileocolitis undergoing recovery; while sometimes the condition passes into an actual ileocolitis. In other severe cases the patient may never recover

from the initial toxic stage, and death may occur in 2 or 3 days, either with severe diarrhea or with obstinate vomiting or both. In other instances the fever is moderate at first, but rises to hyperpyrexia just before a fatal termination. In general it may be said that the severity of the attack is not necessarily in proportion to the frequency of the diarrheal stools. There is always danger that at any time mild instances of the disease may suddenly assume a very severe form and the patient fail rapidly. In all cases in infancy there is constant likelihood of relapse in spite of care, but generally due to some discoverable error in diet. The patient, who appears out of danger, all symptoms having ceased, may then experience sudden recurrence of the disease in a mild or severe form.

The prognosis is, on the whole, favorable in older children, but in infants always uncertain, owing to the various dangers which threaten. It is worse in proportion to the youthful age of the patient, the severity of the symptoms, the existence of previous ill-health, the unhygienic conditions which obtain, and perhaps especially the character of the weather. The sudden onset of exceedingly hot weather may rapidly carry off a patient who had been doing well. Among the poor the disease is more fatal than among the well-to-do, and in hospital practice the course is liable to be prolonged and the prognosis then becomes unfavorable in spite of every care. Breast-fed children not only develop the disease much less often, but offer a vastly better prognosis.

2. ACUTE MILK-POISONING; ALIMENTARY INTOXICATION.—This form of the disease shades into the variety just described. It may occur in infants previously well, but oftener it is a later stage of an intestinal indigestion. It is the "alimentary intoxication," the fourth form, of Finkelstein's classification (p. 698) which is very probably dependent upon an *acidosis*. The cause is apparently clearly a fault in metabolism depending upon the nature of the food; since in favorable cases symptoms cease when nourishment is withdrawn. These symptoms have already been described to some extent (pp. 636, 699). The earliest is fever, either moderate or high; generally accompanied by a diarrhea of various kind and degree, with rapid loss of weight and frequently with vomiting. In well-marked cases there are, further, great prostration; restlessness; lassitude; delirium; coma or convulsions; sunken eyes; shrivelled skin; pinched features; collapse; painful muscular contraction; albuminuria with casts; mellituria; dyspnea with deep respiration; a high degree of leucocytosis; and, in some instances cough. Some cases differ in that there is no fever or diarrhea. This is particularly true of marantic infants. The principal characteristic of the disease is the great predominance of the nervous and other symptoms of acidosis seen from the beginning; the degree of vomiting and diarrhea by no means accounting for the severity of the condition.

Course and Prognosis of Acute Milk-poisoning.—In mild cases, seen early and promptly treated, recovery may occur; but, as a rule, in the severer cases the prognosis is most unfavorable. No change for the better occurs and death takes place in a very few days.

3. CHOLERIFORM DIARRHEA.—This may occur in infants and then is designated by the title *cholera infantum*. In older children it is called *cholera nostras*, constituting the complex of symptoms described by the older writers under the title of "cholera morbus." Both are a severe form of acute gastroenteric intoxication.

Symptoms.—*Cholera infantum* is by no means a common disease, and the great majority of cases to which the title is applied are incorrectly designated. As a rule the attack is preceded by indigestion, or by acute gastroenteric intoxication of the ordinary type; after which the choleric form symptoms set in with great suddenness. Vomiting occurs and is uncontrollable, or, if ceasing for a time, recurs promptly if any food is ingested. The vomited matter is at first the contents of the stomach and then merely a greenish liquid. Simultaneously, or a trifle later, diarrhea develops. At first this is of the character seen in ordinary cases of acute gastroenteric infection, but very soon becomes watery, of a greenish color, and then almost colorless, with but little odor. The stools are very frequent and generally large; or sometimes small and occurring every few minutes and finally involuntarily, the fluids of the body being rapidly lost in this way. Extreme prostration, loss of weight, sinking and filminess of the eyes, shriveling of the face, and pallor and wrinkling of the skin develop with an astonishing rapidity. The whole aspect of the face is completely changed in a few hours. The temperature is usually elevated and hyperpyrexia is common in fatal cases. The pulse is weak and rapid; the respiration often irregular; the urine is nearly or quite suppressed, and may be albuminous; thirst is very great; the abdomen is shrunken; the tongue coated, or red and dry. Often the body becomes cold and cyanotic as in the algid stage of Asiatic cholera, although the rectal temperature at the time may show an elevation of 106°F. (41.1°C.) or over. In other cases the rectal temperature is finally subnormal. Nervous symptoms are marked. At first there is usually irritability and restlessness; later there may be a state of apathy or stupor, or coma and convulsions may develop.

The *cholera nostras* of older children is likewise not a common condition. After some indiscretion in diet there develop very suddenly severe and almost constant vomiting, abdominal pain, and repeated diarrhea. The stools are colored or nearly colorless, and may be extremely offensive. The temperature is elevated; the cerebral symptoms less marked than in infants, but the same wasting and general appearance develop.

Course and Prognosis of Choleric form Diarrhea.—The prognosis in cholera infantum is serious and the majority of cases die. The disease may last not over 24 hours or perhaps 2 to 3 days. In some instances the diarrhea and vomiting continue until death occurs in collapse. In others there may be an abatement or even a cessation of the gastroenteric symptoms, but nervous manifestations with prostration may persist, and death may take place from convulsions or in coma. This is the condition described as pseudomeningitis or hydrecephaloid. In the case of older children the prognosis is likewise serious but not to so great a degree.

Complications and Sequels of Acute Gastroenteric Intoxication.—In the ordinary type of the disease, if at all long continued, complications may arise. Bronchopneumonia and otitis are common, furunculosis and multiple abscesses may develop. Urticaria and erythema are oftener seen in older children; forms of stomatitis are sometimes met with, and renal involvement is not infrequent. Sclerema is a complication reported in cholera infantum, but must be rare in this country. Ileocolitis is a not infrequent sequel. In cholera infantum and in acute milk-poisoning the course is so rapid that there is scarcely opportunity for complications to develop.

Diagnosis.—The diagnosis at the outset is not readily made from acute intestinal indigestion, or from vomiting and diarrhea symptomatic of the onset of acute contagious fevers. *Acute intestinal indigestion* generally exhibits a lower temperature, a shorter course and much less serious involvement of the nervous system. The clinical differences are, however, as stated, only those of degree. Often *ileocolitis* cannot be readily distinguished, especially as it is so frequently a sequel to gastroenteric infection. It generally exhibits a greater amount of mucus in the stools, which are more numerous and smaller, may contain blood, and are passed with pain and frequent straining efforts; while the temperature continues elevated instead of falling in a few days. Only the course of the case can distinguish the symptomatic gastroenteric manifestations which so frequently usher in various *acute febrile diseases*, and sometimes in the intestinal form of *grippe* the diagnosis may be difficult throughout. The differentiation from typhoid fever in infancy is at times difficult. In most cases of acute gastrointestinal intoxication, however, the course is shorter and the fever falls in a few days, and in addition the test for the Widal reaction will be an aid. The nervous symptoms of the severer cases of gastroenteric intoxication sometimes suggest *meningitis* at the outset. Usually the prompt development of diarrhea, uncommon in *meningitis*, makes the diagnosis plain. *Cholera infantum* will be recognized by the violent repeated vomiting and the serous discharges; and the *choleric form* diarrhea of older children could resemble *meningitis* only in the vomiting; its onset, too, being usually much more sudden. In older children *acute poisoning* by arsenic or similarly acting drugs could produce symptoms suggesting *choleric form* diarrhea.

Treatment of Acute Gastroenteric Infection. Prophylaxis.—This is of extreme importance. Weaning must be discouraged, especially in summer time, unless it has been proven beyond doubt that artificial food is necessary. The longer the infant can be successfully breast-fed the less is the danger of gastrointestinal disturbance. The mother's nipples and the infant's mouth must be kept clean, and absolute regularity in the hours of feeding followed (see p. 85). In the case of bottle-fed infants every precaution must be taken to preserve an aseptic condition of the bottles and nipples and of the food employed (see p. 135). Great care must be taken, too, in starting an infant on artificial food, to employ at first very weak mixtures and to increase the strength gradually, feeling one's way carefully as to the special ingredients of the diet which should be increased in amount, and at once checking this increase if signs of indigestion occur. It must be remembered in this connection that mere failure to gain weight is not a necessary indication for adding to the strength of the nourishment.

During periods of unusually torrid weather a temporary decided reduction of the strength and of the amount of the food and the frequency of feeding is an excellent prophylactic measure. The problem of the supply of proper food to bottle-fed infants among the poor in cities, especially during the summer, is a vital one, only to be solved in the charitable supplying of suitably modified milk in such a manner that it can be preserved from bacterial changes after it is delivered to the patients. The civic efforts to instruct mothers in the proper manner of feeding their infants, to which so much attention has been given of recent years, and which has already borne good fruit, is also a matter of the greatest importance. Even in better social conditions, where the best milk can be obtained, it is advisable to pasteurize during the hottest

weather, since, than milk, there is no substance which supplies a better culture-medium for the growth of bacteria. In the case of older children, especially in summer, care must be taken that no indigestible food, such as unripe fruit, is allowed.

The sustaining of the digestive power of the patient by prompt removal from the city during the hot weather is a matter of much importance. The infants of the poor should be sent for the day into the parks, to the sea-shore, or on the river if these are accessible. The infant should be bathed every day, and in very hot weather, where the child shows signs of exhaustion, several short immersions in the tub daily are sometimes of great help in preserving the health. The clothes must be carefully adapted to the state of the weather. Chilling of the surface undoubtedly predisposes, but even to a greater extent does the use of too warm clothing in summer time. Constant supervision of the children of the poor by district physicians and visiting nurses is of great value in the prevention or early recognition of digestive disorders.

1. Treatment of the Attack. ORDINARY TYPE. *Diet.*—Dietetic treatment is by far the most important. This is similar in nature to that recommended for acute gastric or intestinal indigestion. On the first evidence of digestive disturbance food must be withdrawn absolutely. This is Nature's own remedy, as shown by the frequent refusal of nourishment by infants and children at this time. There is no question but that many an infant would be spared a severe attack if no food whatever were urged or even permitted until appetite returned. Water must be offered freely, since what may appear to be appetite for food is often only thirst. Infants at the breast are best given only water, thin barley water, or strained broth for 24 hours, or even longer if severe symptoms persist. After this period nursing may be cautiously resumed, curtailing the duration of each nursing and perhaps alternating with barley water. In the case of bottle-fed infants the same method of treatment should be followed, except that the return to a milk-food must be delayed longer; broth, albumen water, or barley water or other cereal decoction taking its place. Only small amounts of nourishment should be given at a time, and less frequently than in health. Where one kind of nourishment is refused and feeding seems advisable, some other may be tried until the child's fancy is satisfied. It is only in the case of infants already marantic that somewhat prolonged starvation can do harm. Not until after several days, when symptoms have nearly or quite disappeared, can a return to milk be made, diluted skimmed-milk, buttermilk, or casein milk being often a useful milk preparation for first use. Both fat and sugar are liable to disagree, although in many instances whey is remarkably useful in spite of its sugar-content. For infants past the nursing period, and for older children, the avoidance of milk is advisable also, giving broths and gruels instead for a number of days. If the condition persists the various dietetic methods recommended for chronic gastric and intestinal indigestion may be employed. (See pp. 725 and 767.) After recovery at any age the great tendency to relapse and recurrence, especially in summer, make it imperative to use much caution in returning to the ordinary food. Indeed, in summer-weather the diet may need curtailing until the hot season is past.

Hygiene.—In addition to care in the diet, the hygiene of the patient must be considered. The measures suggested for prophylaxis apply here as well. The child should be kept at rest in bed or in its coach, but it is a mistaken idea that patients with the acute symptoms of the disease

must be necessarily confined to the house, although under some circumstances it may be better that they shall be during the excessive heat of mid-day. Abundant fresh air is needed with light clothing if the weather is hot, but, on the other hand, chilling of the surface of the body by cool draughts is to be avoided. During hot weather, or if there is much fever, the use of baths is of value. For this purpose the warm tub bath of 100°F. (37.8°C.), given several times daily, is often better borne and more efficacious than sponging with cooler water. Very important often is a change of climate to a cooler and more bracing region.

Medicinal and Local Treatment.—This is entirely symptomatic. If the case is seen early or if feeding has been persisted in by the mother, a purgative should be administered. Castor oil is excellent, but only if vomiting has ceased. Calomel with soda is a serviceable remedy, giving $\frac{1}{10}$ grain (0.0065) of the former, with 1 grain (0.065) of the latter, hourly or half hourly, to a child of 6 months until 5 to 10 doses have been taken. Milk of magnesia is also an excellent preparation, alone or following the calomel. For older children the solution of citrate of magnesia, 2 to 4 ounces (59 to 118) is useful. In addition to the purgative, irrigation of the colon with a normal salt solution is advisable in nearly every case in order to empty this part of the intestine promptly. Later it may be done once or twice a day while active symptoms last. It should not be continued too long, since the procedure itself is sometimes a source of moderate intestinal irritation. Cool irrigation, too, is often a useful means of reducing fever, if this is unduly high. The employment of the tub bath for the reduction of fever has already been alluded to. When vomiting is persistent and there is reason to believe that the stomach still contains undigested food, lavage with a 1 per cent. solution of bicarbonate of soda is of great service. In place of this the exhibition hourly of $\frac{1}{2}$ dram (2) each of liquor calcis and aqua cinnamomi is an excellent procedure, somewhat larger doses being given to older children. In some cases of vomiting bismuth with bicarbonate of soda is of value. Should diarrhea persist to a moderate degree, not too great haste can be permitted in efforts to check it. This is especially true if fever or nervous symptoms continue; since these may be a sign that there are still irritating substances present. Under such circumstances the administration of castor oil, magnesia, Rochelle salts, or other purgative once or twice daily for a few days is often of value.

After the general symptoms have disappeared a continuance of diarrhea may call for direct treatment. Bismuth is now useful, 5 or 10 grains, (0.32 or 0.65) being given every 2 hours to a child of a year or less, preferably either the subcarbonate or subgallate being employed. The salicylate of bismuth is often prescribed on the ground that its antiseptic power is greater. There seems, however, little reason to believe that the small amount of antiseptic contained could have any real influence on the relatively very large mass of the intestinal contents. Astringent remedies such as tannalbin and tannigen, or the older preparations containing tannic acid, are to be reserved for cases which pass into a subacute stage, the result of the development of ileocolitis as a sequel. Opium is an invaluable remedy in some cases but a dangerous one in others. To avoid it entirely is as much an error as to use it improperly. It must not be exhibited in the early stages when vomiting and nervous symptoms are present, or where there is reason to believe that the locking up of the bowels would be harmful. Later, however, it often happens that there is a too great outpouring of liquid into the intestinal canal and too ener-

getic a peristalsis, as a result of which food and liquid are hurried on and out of the intestine, with consequent prostration and emaciation. In such instances opium is more serviceable than any other remedy. The dose to be employed varies with the case and the effects produced. (See Table of Doses, p. 000.) It is also useful to allay the severe colic which is sometimes a symptom. On the other hand, given inopportunely, it may increase gaseous distention by checking peristalsis too greatly. Colic and distention may, indeed, often be relieved by the application of spice poultices or of stupes, or by the employment of a rectal tube or a small enema.

As the disease in the severer cases is a rapidly debilitating one, measures must be taken to sustain the strength of the patient. Hot mustard baths or packs will be of service if collapse is threatening. Alcoholic stimulation may be needed throughout in the severer cases. The amount to be given and the frequency of dosage varies with the age and the demands of the case, from $\frac{1}{4}$ to $\frac{1}{2}$ dram (1 to 2) of whiskey or brandy being administered every 2 or 3 hours, for a short period only, to a child of from 1 to 2 years of age. Digitalis, caffeine or camphor may be required at times, the last two often preferably given hypodermically. When there has been great loss of fluid from the system, and when vomiting precludes the administration of sufficient liquid by the mouth, enteroclysis is often of value, a warm normal salt solution being given slowly. Only in the worst cases is hypodermoclysis necessary. The procedure is painful and tedious and sometimes an intraperitoneal injection is to be preferred.

2. ACUTE MILK POISONING.—The first indication for treatment is the absolute withdrawal of all food. Nothing but water should be administered, given either by the stomach, if it can be retained, or in the form of normal salt or a 1 per cent. bicarbonate of soda solution by enteroclysis. As the intoxication is dependent upon an acidosis, full doses of alkalies are needed when the symptoms are decided; given either by the mouth, rectum, hypodermically, or intravenously. (See Acidosis, p. 635.) Inasmuch as actual symptoms of acidosis in this disease are liable to terminate fatally, it is well to anticipate this by giving from the beginning sufficient soda to keep the urine alkaline. Hypodermoclysis or intraperitoneal injection with normal saline solution may be needed in some instances. Return to food must be made very cautiously. Other treatment is symptomatic and similar to that recommended for cases of the ordinary type.

3. CHOLERIFORM DIARRHEA.—The treatment of this form of acute gastroenteric intoxication, either in infants or in older children, is necessarily modified by the peculiar character of the symptoms, the extremely rapid course of the case, the frequent persistence of uncontrollable vomiting, the great loss of liquid from the system, and the profound prostration. Lavage of the stomach and intestine should be used at once, cathartics being too slow in their action and being liable to be rejected by the stomach. Morphine hypodermically, in doses of $\frac{1}{100}$ grain (0.0006) to a child of 1 year, repeated in 2 hours if needed, is the sheet-anchor, except in those cases where vomiting and diarrhea have abated but stupor and other cerebral symptoms persist. In these alkaline treatment is indicated. Alcoholic stimulants should be administered freely and frequently by the mouth if vomiting permits of this. Camphorated oil (1:10) and suitable preparations of digitalis, strophanthus, and caffeine given hypodermically, are of service to combat the cardiac

weakness. The excessively high temperature may be modified by cool baths; or when the surface of the body is in the algid state but the internal temperature high, by colonic lavage with cold water. In some cases cold compresses frequently changed may be applied to the head and body while a hot-water bag is kept at the feet; and in the algid stage hot baths (105°F.) (40.6°C.) with or without mustard, or a strong hot mustard pack is of value. Food must be even more strictly avoided, if possible, than in the ordinary type of gastrointestinal intoxication. It is especially in the choleric form type of the disease that hypodermoclysis or intraperitoneal injection should be tried, since liquid given by enteroclysis will not be retained. After the more acute symptoms have subsided the treatment is that described for the milder form of the disorder.

ACUTE ILEOCOLITIS

(Enterocolitis; Follicular Enteritis; Dysentery; Inflammatory Diarrhea)

Considerable confusion has arisen in the description and classification of forms of this affection. The title has been used contradictorily to describe, on the one hand, a class of cases which may exhibit identical clinical symptoms; yet with entire variance in their etiology and pathological anatomy; and, on the other hand, those which may be similar etiologically or present similar lesions and yet be quite different in clinical characteristics. Some of the specific germs, for instance, may be found to be the apparent cause in cases in which the symptoms do not differ from those present when no such germs are discoverable. There is also no sharp boundary-line between this disease and acute gastroenteric intoxication, the lesions seen in this latter being those occurring in the first stage of the former; while clinically the subacute cases of intoxication are not to be distinguished from the milder instances of ileocolitis. The term ileocolitis properly designates a group of cases in all of which the element of *inflammation* is predominatingly present.

Etiology.—Numerous *predisposing factors* are of importance. Children under 2 years of age are especially liable to suffer from the malady, but it is by no means confined to this period. Acute gastroenteric infection frequently has ileocolitis following it, and the disease may readily develop in atrophic children the subject of chronic intestinal indigestion. It is, too, a sequel at times in older children to some of the acute febrile diseases, such as measles, typhoid fever, pneumonia and diphtheria. It may, however, readily, and perhaps oftenest, occur as an acute primary disease not preceded by any digestive or other disorder.

Summer time witnesses the majority of cases, dependent not only upon the direct influence of hot weather on the system, but upon the ready contamination of the food by microorganisms at this season. Lack of satisfactory hygiene of any sort predisposes to the disease. Consequently, as in the case of other digestive disorders in infancy, it occurs oftenest in crowded districts in cities, and even in well-regulated institutions where many infants are maintained. Epidemic influence is very positive. This is especially true of tropical countries, but there may be local outbreaks in temperate regions. It is oftenest, however, seen sporadically. Whether it is contagious from one child to another, or whether several children ill together may have been infected from some outside source is not yet definitely known, but the latter seems more probable.

Ileocolitis is, however, a distinctly infectious disease and the *exciting cause* is a germ of some sort. In tropical dysentery this may be the

ameba coli, so named by Lösch,¹ and studied especially by Kartulis.² This microorganism is only occasionally met with in temperate climates; and this is especially true of children. In over 3000 children seen by DeBuys,³ only 4 had amebic dysentery. Several different species of bacteria appear to be able to produce the disorder, among them being the colon-bacillus, a variety of streptococcus, and the bacillus pyocyaneus; but the germ which seems to be perhaps oftenest the cause is the dysentery-bacillus. This was first described by Shiga⁴ in 1898; and shortly afterward, in 1900, Flexner⁵ reported the discovery of another strain, differing from the Shiga-type in that it produced in culture an acid reaction. The presence of this variety in ileocolitis, as well as in other diarrheal conditions in children, was observed by Duval and Bassett⁶ in 1902. Different strains of this germ have been described. The original Shiga, or alkaline, type, is uncommon in infants in this country, and much the more numerous cases are associated with the acid type of the germ, the Flexner variety, occurring alone or sometimes accompanied by other strains, as, for instance, that of Hiss and Russell.⁷ In the Collective Investigation of the Rockefeller Institute⁸ in 1904, 412 infants with diarrheal disease were studied, and the dysentery bacillus found in 279; *i.e.* 63.2 per cent. Of these only 29 cases showed the presence of the bacillus of the Shiga type.

That the dysentery bacilli are in reality the cause of many cases of diarrheal disease in children would seem to be indicated by the fact that an agglutinative reaction develops with the blood taken from the patient.

It is important to remember, however, that not only is it not proven that the dysentery bacillus is the sole cause of ileocolitis in children, but it is certain that these germs may be present in other diarrheal conditions, and even very exceptionally in the stools of normal infants. In some cases of ileocolitis an agglutinative reaction has been found with the streptococcus (Jehle).⁹ The number of bacteria present in the stools is generally comparatively small, but to this there are exceptions. The mode of entrance into the body is unknown. The germs are capable of living but a short time outside of the body, but probably may continue to exist and to propagate themselves in the intestine for long periods after the disease itself is over.

Pathological Anatomy.—The lesions vary greatly in situation, kind, and intensity, dependent to a large extent upon the duration and severity of the attack. The basic character of the disease is *inflammation*, this distinguishing it from the diarrheal disorders already described. As regards situation the large intestine is the most frequent seat, the lower part of the ileum being much less affected and with more scattered and superficial lesions; but even where most abundantly developed the lesions are not uniformly distributed, parts of the intestine being greatly involved and the neighboring parts to a much less extent if at all. The degree of change produced varies with the case; and, based upon the post-mortem lesions, the disease has been divided into different types. In the **acute catarrhal form** the mucous membrane is congested and swollen, often

¹ Virchow's Archiv., 1875, LXV, 196.

² Centralbl. f. Bakt., 1891, IX, 365.

³ Journ. Amer. Med. Assoc., 1914, LXIII, 1806.

⁴ Centralbl. f. Bakt., 1898, XXIII, 599.

⁵ Johns Hopkins Hosp. Bullet., 1900, XL, 231.

⁶ American Med., 1902, IV, 417.

⁷ Medical News, 1903, LXXXII, 289.

⁸ Bact. and Clin. Studies of the Diarrheal Diseases of Infancy, 1904, 124.

⁹ Jahrb. f. Kinderh., 1907, LXV, 40.

covered with mucus, and the epithelium loosened in places. Small hemorrhages, usually scattered or in streaks, are seen on the surface of the mucous membrane especially upon the projecting portion of the folds, and superficial erosions may be found; and in severe cases these shallow ulcerations may be extensive, and the whole intestinal wall may appear much thickened. Both the solitary and the agminated follicles are generally swollen and the villi are elongated and prominent. The change in the agminated follicles may closely resemble the appearance in typhoid fever. Microscopically there is found an infiltration of small cells and of very numerous bacteria of different kinds in the mucous layer, penetrating even to the muscular layer in severe and long-continued cases. The lymph-follicles are infiltrated. In cases which recover the lesions disappear entirely.

If the diseased process has advanced further, **follicular ileocolitis** is also found. There is here a more or less deep ulceration in the solitary follicles. These ulcers are at first very minute, but by fusion with each other and extension into the adjacent mucous tissue they may produce lesions of considerable size with overhanging walls. There is a moderate infiltration of the submucous layer, and in severe cases the muscular layer is much thickened and infiltrated, and the ulceration extends into it. Cases which recover do so with cicatrization of the ulcers.

In the most severe form of the disease a **membranous ileocolitis** develops. In this condition there are regions in which the entire thickness of the intestinal wall becomes much swollen and stiff, with an obliteration of the usual folds, resulting from the presence of fibrinous exudate and infiltration by round cells. The surface is rough and has not the ordinary appearance of mucous membrane, and the various structures constituting it are not readily distinguishable from each other. In other regions where the membranous deposit has become detached, deep ulceration may be seen.

In general, acute catarrhal inflammation is found in the milder cases of short duration; follicular inflammation does not usually develop until in the 2d week; membranous inflammation is usually attended by severe symptoms, and may occur even comparatively early in the disease. But there is no certain relationship in ileocolitis between the severity of the symptoms and that of the lesions. Cases with extensive ulceration, for instance, may sometimes exhibit but moderate fever and little or no blood in the evacuations, while catarrhal inflammation may perhaps be attended by bloody mucous stools, and the case may continue for some weeks and end fatally without any follicular ulceration having developed.

Lesions of other regions are often associated with those of the intestine. Bronchopneumonia is not infrequent and swelling of the mesenteric glands may be found. Degenerative changes in the kidneys are often seen and occasionally true inflammatory alterations as well. Enlargement of the spleen may occur, with degenerative changes in it and in the liver and other organs.

Symptoms.—For reasons already pointed out a division of the disease into clinical types based upon pathological and bacteriological findings is possible only to a limited extent, and the symptoms vary greatly. In what might be called an *average* case there may have been an earlier simple diarrhea or gastroenteric intoxication, which gradually merged into or suddenly developed a condition of ileocolitis; or the disease may start abruptly as a primary affection, perhaps following an indiscretion in diet, with practically no prodromes. The onset cannot be

distinguished with certainty from that of other acute intestinal disturbances. In the more acute cases there is fever of from 103° to 104°F . (39.4° to 40°C .), often vomiting, abdominal pain, and diarrheal movements containing undigested food. Very promptly the stools become very frequent, small, are passed with straining efforts, and exhibit mucus either transparent or green in color, pus, fecal matter, and more or less blood. The abdomen becomes moderately distended and somewhat tender; the urine is scanty and may contain albumin. There is loss of appetite,

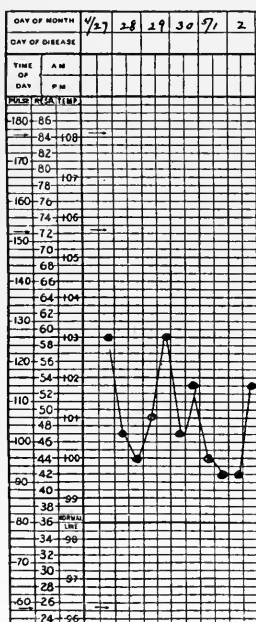


FIG. 250.

FIG. 250.—ACUTE ILEOCOLITIS OF MODERATE SEVERITY.

Mollie G., aged 7 months. Bottle fed. Apr. 27, been ill 11 days with mucus, blood and pus in the movements, very frequent, with straining, fever, prolapse of the rectum. Condition now apparently improving slowly. Temperature uncharacteristic.

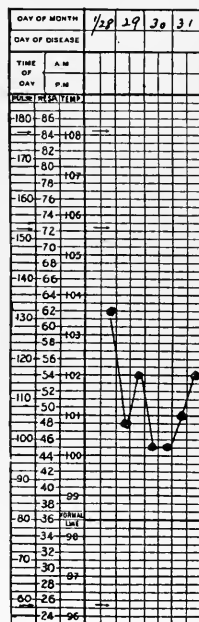


FIG. 251.

FIG. 251.—ACUTE ILEOCOLITIS.

Arcteso P., aged 6 months. Illness began with fever, vomiting and diarrhea, with green, mucous stools, a week before admission to the Children's Hospital of Philadelphia, Jan. 28. Child weak, looking ill, cyanotic, having 5 to 8 green, mucous stools in the 24 hours, with a little blood. Death on the 31st. Temperature uncharacteristic.

coated tongue, thirst, decided prostration and rapid loss of weight. The appearance is that of an ill child. The tenesmus is not constant and the abdominal pain is colicky in nature; both symptoms developing or increasing at the time of evacuation of the bowels. Prolapse of the rectum is not uncommon.

In the *severer* cases evacuations may be accompanied by great tenesmus and colicky pain, may occur every hour or even much more frequently, and may contain blood-tinged mucus only, with little or no fecal matter. The tongue is dry; sordes appears on the lips and teeth; there is great restlessness or apathy, prostration, sinking of the eyes, stupor, and sometimes delirium or convulsions. In *milder* cases char-

acterized by slower onset the stools contain a considerable amount of undigested food in addition to mucus, the straining and abdominal pain are less marked, and other constitutional symptoms are but little developed. Sometimes the odor of the stools is very offensive, but this is less apt to be the case when mucus is their chief constituent.

The temperature in ileocolitis is very variable. It is liable to rise rapidly in the primary and the severer cases and continue high; or to become less elevated than at the onset and more irregular in the milder cases; but this is open to numerous exceptions (Figs. 250 and 251).

Course and Prognosis.—The duration and development of symptoms varies with the intensity of the attack. In very severe cases the bloody, mucous character of the stools continues unaltered, their frequency undiminished, fever remains high, prostration and loss of weight increase rapidly, and death takes place in from a few days to 1 or 2 weeks. In other severe cases the toxic cerebral symptoms occupy the most prominent position, or are combined with intestinal manifestations; there being vomiting, delirium, unconsciousness, prostration, and convulsions. Such cases may exhibit few symptoms considered characteristic of dysentery. In cases of *catarrhal ileocolitis of average* severity the stools, which were at first almost entirely of glairy mucus with blood, exhibit more or less increase of fecal matter after 2 or 3 days, are nearly odorless or very offensive, and become more fluid and greenish or brownish, while blood nearly or quite disappears, although abundant mucus is still present. The high temperature of the onset becomes lower and more irregular, and decided loss of flesh and strength continue and is sometimes very great. Then after 1 or 2 weeks in favorable cases improvement begins, pain and tenesmus lessen, the stools grow decidedly less in number and are more fecal and with less mucus, the temperature diminishes, and strength increases. Convalescence is, however, slow, and it may be 4 to 6 weeks before mucus has disappeared and recovery is complete. Relapses are meantime very prone to occur. These may develop after convalescence seems established, or there may be a return of severe symptoms, with blood and increase of mucus at various times during the course of the attack. Should the disease prove fatal the symptoms persist or increase in severity, prostration grows more marked, the appetite is lost, the tongue dry, the evacuations continue to exhibit mucus and blood, there may be occasional vomiting, delirium or convulsions may appear, and death occurs after 4 to 6 weeks from exhaustion or from some intercurrent condition, especially bronchopneumonia. In all the cases lasting several weeks it is probable that the disease has reached the stage of ulceration. Such ulceration always renders the prognosis more unfavorable.

In the *mild catarrhal* cases the fever is never high, the stools not very numerous and unaccompanied by much pain or straining, there is little prostration, and recovery may take place in from 10 days to 2 or 3 weeks.

In the cases of *follicular ileocolitis* the condition may follow the acute catarrhal form, or very frequently be a sequel to an acute gastro-enteric intoxication, the symptoms of the latter merging gradually into those of the former, and the case being of a rather subacute type from the beginning. Whether or not the onset in this form is sudden, the temperature soon grows irregular and seldom high; the evacuations contain both mucus and fecal matter, little if any blood, are not very numerous, and are passed with no excessive straining; marked nervous

disturbances are absent; prostration is not great at first; emaciation is progressive and constant and finally becomes extreme, and death usually results in 3 or 4 weeks or longer, with the symptoms of advanced malnutrition. Improvement in such cases is an indication that the follicular ulceration has not become extensive. Recovery is always very tedious and relapses very frequent. These may be brought on by any indiscretion in diet, chilling of the body, or any departure from careful hygiene.

In general it may be said that the prognosis of ileocolitis is much graver if ulceration has developed, but that the determining of the existence of this condition usually cannot be made with certainty, the presence of blood generally depending upon congestion rather than ulceration. If the disease has lasted only a week or less and improvement in the evacuations then begins, it is likely that there is no ulceration. If there is no improvement in 3 or 4 weeks ulceration is very probably present. Between these two there is a middle ground where the presence of ulceration is only conjectural. The prognosis is also influenced by the age, being worst in infancy; while any debilitating disease or impaired state of health is also unfavorable. The primary cases give a better prognosis than the secondary ones. The season of the year is of great importance, hot summer weather making recovery much more difficult and relapse especially likely to occur. Any unfavorable hygienic surroundings increase the danger. Hence in cities and among the poor the disease is more dangerous, as it is also in hospitals and other institutions in spite of every effort for the maintenance of proper hygiene. The prognosis of ileocolitis is always serious and uncertain, and the death-rate is high.

Complications and Sequels.—Bronchopneumonia is a frequent and serious complication, appearing generally late in the course of the case. Thrush or other forms of stomatitis may develop. Sepsis is a frequent and early cause of death in severe cases. Degenerative lesions of the kidneys are common. Purulent otitis, or furunculosis and other cutaneous suppurative conditions, may occur. The most important sequel not infrequently following is a chronic ileocolitis. In other cases a persistent malnutrition may remain for months after the disease itself is over. Obstinate constipation is a not uncommon sequel.

Diagnosis.—The principal diagnostic symptoms of the disease are the tenesmus, abdominal pain and tenderness, fever, and frequent, small stools containing mucus and more or less blood. Yet the symptoms vary to such an extent that the diagnosis is often not easy. When preceded by *acute gastroenteric infection* it is often impossible to determine just when the actual inflammation began. The development and persistence of fever and pain and the constant occurrence of mucus in the stools render the existence of inflammation probable. *Intussusception* may simulate ileocolitis very closely at the onset; both conditions exhibiting tenesmus with the passage of bloody mucus. The former is, however, characterized by the more sudden onset without initial fever, the complete lack of fecal matter in the stools, the absence of the passage of gas from the bowels, the more rapidly developing prostration, and the discovery of a tumor through the abdominal walls or by rectal palpation. *Typhoid fever* in infancy exhibits vague symptoms and often much resembles ileocolitis; and indeed the pathological lesions are often much the same. The onset in typhoid fever, however, is usually less abrupt and there is no tenesmus, while the Widal reaction and the roseola are

characteristic. Enlargement of the spleen may occur in either disease, but is more common and more marked in typhoid fever. Some of the severer cases of ileocolitis with minor intestinal symptoms and marked cerebral ones may readily suggest *meningitis*. Although diarrhea of any degree is a contra-indication to the presence of this, yet this statement is by no means invariably true, and the diagnosis may be one of great uncertainty.

The obtaining of an agglutinative reaction with the blood of the patient is a diagnostic test of but little value. It does not appear before the 2d week, and is "not an index of the presence of, or infection with, *Bacillus dysenteriae*" (Flexner).¹

So far as any differentiation clinically can be made of the different forms of ileocolitis from each other, *catarrhal* inflammation is indicated by the more sudden onset and the greater amount of mucus and blood in the stools, with tenesmus and abdominal pain. In the milder cases of this form the temperature soon diminishes and the mucus and straining grow less. If superficial ulceration, not follicular, is present the course is longer. *Follicular* ileocolitis is usually secondary; the onset is often less abrupt; the stools contain more fecal matter and little blood, although always much mucus; tenesmus and pain are but slightly developed; the temperature is lower or more irregular, and the course is subacute. The *membranous* variety has no certain characteristic symptoms except the possible discovery of shreds of pseudomembrane in the stools. Its onset may be sudden; the temperature continuously high; the nervous manifestations, such as delirium, marked. The mucus and blood in the stools and the character of the disease in general may suggest a severe case of the catarrhal form of the disease. In other instances, however, the intestinal symptoms are vague and the constitutional ones, and especially those of a cerebral nature, misleading and lead to the mistaken diagnosis of a meningitis.

Treatment. Prophylaxis.—The same methods of management are to be followed as described under Acute Gastroenteric Intoxication, every attention being given to the maintaining of the general health, the escape from excessively hot weather, and especially the guarding against indigestible food or that containing bacteria in excess. The existence of other diarrheal diseases being a powerful predisposing factor, such must be treated as thoroughly and as promptly as possible. In addition is to be borne in mind that ileocolitis is an infectious disease and that the spread by contagion, although probably not a factor of moment, is at least a possibility, and precautionary measures may well be taken against this.

Treatment of the Attack.—Here the first matter to be considered is the *diet*. Early in the disease milk should be withdrawn completely, and this applies even to breast-fed infants. In place of milk such articles should be given as broth free from fat, and barley water or other thin cereal decoction except oatmeal, frequently and in small amounts. After a day or two breast-feeding may be resumed; but in the case of artificially fed infants a somewhat longer delay in returning to milk is advisable, until the acute initial symptoms subside. The strength of the food must now necessarily be increased, since so often appetite is poor and not sufficient nourishment is taken. Among foods suitable at this time are stronger cereal decoctions in the form of gruels, which sometimes

¹ Bacteriological and Clinical Studies of Diarrheal Disorders of Infancy, Rockefeller Institute, 1904, 135.

may be dextrinized with advantage, but with caution lest diarrhea is increased thereby; egg-water; beef-juice and broths, the latter to be thickened with starchy addition and often containing the animal fibre in finely divided form. Scraped rare beef is serviceable in older infants. The return to milk must be made with great caution, and this at once withdrawn if it is passed largely undigested or increases the symptoms. Whey; skimmed milk, perhaps peptonized; casein milk, and butter-milk are often of great value. Only trial can determine the food best suited to the case. The fat of the milk is often badly borne for a long time. Although food should be given frequently and in small amounts, it must not be administered too often lest the intestinal peristalsis be increased. In many cases the intervals between feedings should be lengthened. Particular care is to be observed that enough nourishment is taken, since the course of the disease is liable to be prolonged. Sometimes the loss of appetite renders feeding by gavage necessary. Water should be given freely, since there is often so much loss of liquid from the intestine. Even when convalescence is advancing the very greatest care in diet must still be maintained, perhaps for some months, since relapses are readily brought about by the slightest indiscretion. In the case of older children all foods of the green-vegetable class, or those with much waste material, such as oatmeal, and all fruits must be carefully avoided.

Hygienic treatment is of importance likewise. Abundance of fresh air is essential yet with the avoidance chilling of the body. The patient should be at rest in bed. Change of locality is often of great value in the later stages of the long-continued cases; and if the weather is hot the patient should at once be removed to the seashore or mountains at any stage of the disease.

Medicinal and Local Treatment.—At the beginning of the attack the child should receive a purgative of castor oil, calomel, or a saline, and the bowels should be well washed out with a douch of normal salt solution. After the action of the purgative two courses are open so far as drugs are concerned. One, the administration daily of castor oil or a saline purgative until the character of the stools changes; the other the giving of bismuth subcarbonate in 6 to 10 grain (0.39 to 0.65) doses every 2 hours at 2 years of age, combined with opium. If there is reason to believe that irritating material has been thoroughly removed from the bowel I prefer as a rule the latter plan, although the former is often very efficacious. The continued employment of lavage of the intestine 2 to 3 times daily with water either warm or cool, according to the effect, is sometimes very successful, using from 1 to 2 quarts (946 to 1892) at a time. Either a normal saline solution or, often preferably, starch water may be used for this purpose. In very many instances, however, enemata after a time increase the irritation and tenesmus and must then be promptly abandoned. The employment of enemata of nitrate of silver, tannic acid, or other astringent is, in my experience, oftener harmful than of benefit, as it is liable to increase the tenesmus or to cause violent resistance on the part of the child. If used at all they should be decidedly weak, not more than 0.25 to 0.5 per cent. of tannic acid or 0.1 per cent. of nitrate of silver.

Pain and tenesmus are to be treated by opium in sufficiently large doses, frequently repeated. The employment of this should be delayed, if possible, until the bowels have been thoroughly evacuated by the initial purgatives and enemata. Later in the case the administration of some

tannic-acid preparation by the mouth, such as tannalbin or tannigen, is sometimes of service, still combined with bismuth. Acetate of lead, sulphate of copper, and nitrate of silver in small doses internally are sometimes employed. Emetine would appear to be a specific in cases of amebic dysentery. As the disease is an exhausting one stimulants are often required early, given in doses proportionate to the age (see pp. 223, 229), bearing in mind the fact that children tolerate alcoholic stimulants in relatively large amounts. Among other remedies used to sustain the strength as the needs demand, are digitalis, strophanthus, caffeine and camphor, best given hypodermically.

Serum therapy, even in cases proven to be dependent upon the dysentery-bacillus, has not appeared as yet to be of any decided value.

During *convalescence* great care must be taken with the diet on the lines already indicated, and chilling and fatigue of the body guarded against, since relapses so readily occur. Tonic remedies are indicated, especially nux vomica and often iron.

CHRONIC ILEOCOLITIS. CHRONIC DIARRHEA

These two disorders are practically the same, any diarrhea which runs a decidedly chronic course being liable to be finally associated with inflammatory lesions more or less well marked, whatever the original cause may have been.

Etiology.—A common cause is an attack of acute ileocolitis which, instead of disappearing, has passed into a chronic form. In other very frequent cases the disease develops insidiously in patients who have been suffering from a diarrhea attending rickets or other chronic debilitating disease, or who have had long-continued chronic intestinal indigestion from persistently faulty feeding.

Pathological Anatomy.—The lesions vary, depending somewhat on the original disease. Only a catarrhal condition of the mucous membrane may be found. To the naked eye there may be little alteration in appearance, except some thickening, with pigmentation of the mucous membrane; not uniformly but in patches or streaks, or seen chiefly in the enlarged solitary glands or in Peyer's patches. Microscopically a decided cellular hyperplasia is found, and there may also be more or less cellular proliferation and atrophy in the epithelial glandular tissue of both the large and the small intestine, with hypertrophy of the connective tissue. In the less frequent, severer cases, especially those following severe acute ileocolitis, there may be decided thickening of the intestinal wall, with ulceration or with evident cicatricial tissue the result of the healing of ulcers.

Associated with the intestinal lesions, changes in other organs are often observed; pneumonia and degenerative alterations of the kidneys and liver, the latter often being fatty, and enlargement of the mesenteric lymphatic glands being among these.

Symptoms.—These are largely those of increasing debility and marasmus, combined with diarrhea and other digestive disturbances. The diarrhea is not usually severe but is persistent or constantly recurring. The stools are looser than normal although not very thin: number perhaps 2 to 6 daily; are large in size; sometimes foamy and always contain more or less mucus, with undigested food, and sometimes pus in small amounts. Blood is not often present. The odor is generally very offensive; the color either dark brown or light brown, or sometimes green-

ish or greyish. The abdomen is commonly distended with gas. Pro-lapse of the rectum may take place, although less often than in the acute cases. Vomiting is not common, fever is absent or occurs only in temporary outbreaks, and colic, abdominal tenderness, and tenesmus may be observed but are not as frequent as in acute cases. The tongue is coated or sometimes dry and red; the appetite is often unaffected or is increased, but sometimes very poor. The general health is greatly affected. The child suffers from irritability or apathy, malaise, disturbed sleep, poor circulation, dry, rough skin, anemia, and emaciation which is sometimes very extreme. There is a very persistent whining cry. A marantic dropsy often develops.

Course and Prognosis.—The course is essentially chronic but is not uniform. Whether death or recovery finally ensues there are liable to be periods of transitory improvement followed by relapse. In other instances a temporary disappearance of mucus from the stools may be attended by the development of fever. In the cases which recover, in which the intestinal lesions were probably of lesser severity, half a year or longer may go by before convalescence can be said to be assured, and even then the general health may not yet be fully regained and the digestion may require careful watching to prevent the relapses which so frequently occur. The fatal cases may exhibit improvement at times, but may grow rapidly worse in some one of the recrudescences, and the patient die from exhaustion or from some complicating intercurrent disorder, not infrequently bronchopneumonia. Toward the end of life an extensive development of petechiæ is not uncommon, especially on the abdomen.

The prognosis in general is always serious, and difficult to formulate for the individual case. The most severe cases, given up by physicians of experience, will sometimes eventually recover. If intestinal lesions are marked the prognosis is very unfavorable; but, inasmuch as we cannot during life know the exact severity of these, an absolutely unfavorable prognosis must be given with reserve. Those patients usually die who possess little general strength, are under unsatisfactory conditions, and in whom the disease has already lasted for some time without any improvement.

Complications and Sequels.—Bronchopneumonia is a common complication. Corneal ulcers are sometimes seen as are purulent otitis and suppurative processes in the skin. Thrush is common, nephritis may be a complication, and tuberculosis a not infrequent sequel.

Diagnosis.—The nature of the disease is usually sufficiently evident in well-developed cases. The milder forms with but little diarrhea cannot always be sharply distinguished from cases of chronic intestinal indigestion. The diagnostic features are the large amount of mucus constantly occurring in the stools, and the presence of the inflammatory characteristics of ileocolitis. It is important to distinguish, too, as far as possible, the nature of the cause, inasmuch as this has such an important bearing upon the prognosis. When the disease follows an acute ileocolitis, if this has been severe and of long duration, it is probable that the case is one of the ulcerative type. If it has developed slowly in debilitated subjects or has followed a chronic intestinal indigestion, there is greater likelihood of it being catarrhal or follicular without ulceration. The distinction from a tuberculous enteritis is often difficult. As a rule, however, tuberculosis of the intestine is a later manifestation of this disorder already evident in other parts of the body, especially the

lungs; the onset is slow; fever is present; there is often blood in the stools and there already exists decided involvement of the general health out of proportion to the severity and the duration of the intestinal symptoms.

Treatment.—The principal treatment is dietetic and hygienic. In the matter of *diet* only trial can determine the food best suited to the patient, the examination of the stools being of aid in this. Peptonized milk preparations are of service in occasional instances, especially in infancy. In others it is better to use skimmed milk, since the fat may be not well tolerated. In other instances casein milk or buttermilk answers well. In very many cases, however, milk in any form is always passed undigested and should be avoided, and broths thickened with a starchy addition are to be preferred. Dextrinized starchy foods are frequently better than unconverted starch; yet in some instances, as with broths, increased frequency of the stools may result. Beef juice, scraped beef and white of egg are often very serviceable.

In the line of *hygiene*, rest combined with abundance of fresh air is very important. Massage is sometimes of value. Precautions must be taken as far as possible against exposure to hot weather, by removing the child to the seashore or the mountains. Indeed, this decided change of air is often the most successful treatment. Large enemata of saline solution or of starch-water are useful for washing out the bowels when the stools are more abundant than usual, or should any temporary constipation occurring be attended by unfavorable symptoms. They should not be given routinely, but with frequent interruptions in order to determine whether or not their employment is keeping up the discharge of mucus from the bowel. Astringent enemata are probably of more service than in the acute cases, but are often irritating.

The giving of *drugs* by the mouth is a very minor part of the treatment. Bismuth or tannalbin may be used on the occasions when the frequency and thinness of the stools is temporarily increased. If this treatment is not sufficient and there is reason to think that peristalsis is too active, opium may be given, but cautiously and not continuously lest constipation develop, with consequent exacerbation of the fever and other general symptoms. A combination of opium and dilute sulphuric acid is useful in some instances. Castor oil or calomel may be employed at intervals if the stools are unusually offensive or if there has been a rise of temperature apparently due to intestinal intoxication, and it is even serviceable without these indications. It may be necessary to administer alcoholic stimulants freely and continuously to support the strength. In some cases the continued administration of cod-liver oil has proved of benefit. I have also seen improvement follow the persistent employment of tincture of the chloride of iron.

CONSTIPATION

Although only a symptom, constipation is one of extreme importance and frequency at all periods of life. By the term is indicated evacuations which are either too infrequent, too small in amount, or too firm and dry. The title is, however, a relative one and is applicable more to the character than to the frequency of the stools, unless this is much diminished, while always the individual habit is to be borne in mind. Some infants, for instance, of an age where 2 to 3 stools should occur daily have regularly but 1, and should not therefore be called constipated if the evacuation is of sufficient size and of normal character. In other cases in older children, where but 1 movement daily is to be expected, there

may be 2 or 3, but these may be hard and small and constipation is present.

Etiology.—The causes are various and numerous, and the etiological factors are not the same at different periods of life. Age appears to have little influence in determining the frequency. In the new born entire absence of bowel-movements may depend upon atresia of the anus or imperforate rectum, or upon complete obstruction higher in the course of the gastroenteric tract. (See *Intestinal Obstruction*, p. 78.) In other instances the condition at this early age is less serious, and a temporary constipation lasting 1 or 2 days at the first results from a lack of tone in the intestinal wall, combined with the fact that no food has been ingested. In other infants, or in older children, constipation may depend upon congenital dilatation of the colon; stenosis of the pylorus or of the intestine at some portion; appendicitis; peritonitis; strangulated hernia; fissure at the anus which causes pain at stool and consequent unwillingness of the child to make the necessary effort; hemorrhoids acting in a similar manner; intussusception, or other forms of intestinal obstruction, including the blocking of the intestines by a foreign body or by a large, hardened mass of feces.

Other agencies, however, are far more frequently active than these mentioned. A predisposing cause of a constipated habit in infants is the unusual length and tortuous course of the sigmoid flexure characteristic of this period, to which attention was called by Bednar¹ and especially by Jacobi.² The character of the diet is a very frequent factor; not uncommonly constipation in infants being due to a deficiency in the quantity of food taken or in the strength of this, or to a lack of a sufficient amount of some one of the ingredients. A breast-milk or a bottle-mixture poor in fat is very liable to produce constipation; one poor in protein does the same to a less extent. Sometimes too fat a food produces constipated soap-stools. In the case of some infants sterilizing the mixture is a cause; in others the addition of barley water or any other similar cereal decoction may occasion the trouble; in still others lime water appears to act in this manner. It is largely a matter of individuality. After the age is passed where the diet should consist solely of milk, constipation may depend upon the persistence in the use of this as the chief food taken; or upon the limitation of the diet to foods which are too digestible, such articles as green vegetables, fruits, and those cereals like oatmeal with considerable waste matter in them having been neglected largely or altogether.

Perhaps the most frequent producer of constipation is a certain degree of atony of the intestinal musculature. Anything which causes general debility, such as rickets or anemia, readily gives rise to this lack of tone by affecting the intestinal muscles and those of the abdominal wall. Convalescence from any disease, and especially from diarrheal disturbances, is likely to be attended by constipation. In all such cases of loss of tone the stools may be of normal consistency and there may be lacking only the expulsive power, but oftener the long continuance in the intestine has been followed by an absorption of the water and the feces are too firm. A common cause of failure of muscular tone is lack of sufficient exercise; and consequently the trouble is more common in children in winter-time from the greater confinement to the house and the long sitting in school. The too frequent employment of enemata or of purga-

¹ Die Krankh. d. Neugeborenen, 1850, I, 64; 128.

² Amer. Journ. Obstet., 1869-70, II, 96.

tives is a fertile source of loss of muscular power, in that this accomplishes artificially what the intestine should do of itself. Nervous influences effect greatly the intestinal muscle. The failure to observe regularity and the ignoring of the sensation of a desire to evacuate the bowel will soon develop a constipated habit. The hurry of the morning hour in school children, haste or other disturbances during the time passed in the toilet, and many similar influences are effective. Heredity is also a nervous factor of importance. With all instances of lack of muscular tone there is liable to be combined deficiency in the secretory action of the intestine and a consequent change of its contents from the normal condition.

Symptoms.—Constipation may be either acute or chronic. The *acute form* may be congenital and persistent, while other severe acute cases may develop dependent upon some of the organic causes mentioned, and attended by symptoms of these disorders. In the milder acute cases there may be no symptoms whatever other than the constipation; or the condition may be associated with evidences of indigestion, colicky pain, and distention, together with slightly toxic symptoms such as headache, torpor, fever, and similar manifestations, which seem often to be dependent upon the blocking up of the bowel, and which not infrequently suggest the onset of some acute febrile disease. The bowel-movements in acute constipation have the ordinary character of the constipated stool. They are infrequent; there may be a desire to evacuate with inability to accomplish it; and when finally a stool is passed after straining efforts it is unduly hard, dry, and large, and perhaps streaked slightly with blood and mucus. The condition may last several days with only unsatisfactory stools or with none at all.

In *chronic* constipation there may be either a constant repetition of acute attacks with short intermissions, or the bowels may be persistently sluggish, requiring always more or less aid to relieve them. Sometimes there is never an evacuation unless artificially obtained; sometimes there may be stools passed without aid, but these are abnormally firm in character. Symptoms may be entirely lacking except the constipated condition, and the child appear in good health in other respects; but frequently there are abdominal pain and flatulence; symptoms of indigestion in general; loss of appetite; occasional fever; sometimes vomiting; anemia; high colored, scanty urine containing indican in considerable amount; and various nervous symptoms, including headache, disturbed sleep, and even convulsions. Not only may the general health be thus affected, but local disturbances may arise, such as fissures, prolapse of the anus, hernia, or hemorrhoids. Not infrequently retained fecal masses may be felt as hard nodules through the abdominal walls, and occasionally these may attain a tumor-like size and hardness.

Course and Prognosis.—In the acute cases the prognosis depends upon the etiology. Relief is obtained in a few days when the disturbance is from acute indigestion and similar removable causes, while in those due to organic obstruction the prognosis is serious. In the chronic functional cases there is no direct danger to life, but the course is indefinite and more prolonged and the general health often suffers seriously. When dependent upon chronic dilatation of the colon the prognosis is more serious.

Diagnosis.—The condition is generally evident from the history. One may sometimes be misled by the statement that a daily evacuation of the bowels occurs, but investigation may show that this is insufficient and that in fact much fecal matter is being retained in the colon with the

consequent development of abdominal distention, evidences of indigestion, and loss of health. The nervous manifestations attending some of the acute attacks or the exacerbations of the chronic condition may simulate so closely the onset of acute infections, including meningitis, that difficulty in diagnosis may at first exist; removed later by the prompt recovery following the administration of a purgative. Fecal concretions in the colon discovered by palpation may sometimes suggest the presence of a tuberculous peritonitis or of morbid growths. A careful examination combined with a study of other symptoms will generally remove all doubt.

The chief object of diagnosis is to discover the cause. In the acute cases this is imperative, inasmuch as only surgical interference can avail for the relief of some of them; but in the chronic cases also it is of importance in order to select the proper treatment.

Treatment.—Attention must be given in *acute constipation* to the immediate unloading of the bowel. Where the constipation is complete and depends upon organic obstruction operative interference is required; when the result of other causes, the emptying of the intestine may be accomplished by enemata of soap and water. If the feces are very hard, 2 to 4 fl. oz. (59 to 118) of cotton-seed oil may first be injected and allowed to remain for a few hours; and if the mass is also large it may be necessary carefully to break it up with the finger or with a smooth-handled teaspoon. In many instances of brief duration a soap-stick is useful in the case of infants, or a glycerine suppository for older children. When evidences of indigestion accompany the constipation a cathartic is more serviceable, using calomel, castor oil, or milk of magnesia in infants, and in older children citrate of magnesia, calcined magnesia, castor oil, or rhubarb.

In the treatment of *chronic constipation* the chief attention must be given to the cause. As this is oftenest the *diet* it may be considered first. In the case of breast-fed infants aid may occasionally be obtained by a modification of the mother's milk through attending to her own diet and hygiene, the effort being made to augment the supply and increase the total solids (see p. 106), if examination shows these to be deficient. In bottle-fed babies some alteration of the diet should be made. The amount of fat may be raised cautiously to 3 or 4 per cent., if this is found to be well digested. The protein should not be too high, as this appears sometimes to favor constipation, but on the other hand it should seldom be less than 1 per cent. or proper development may be interfered with. In some instances the use of oatmeal water or of bran-water as a diluent is of benefit. Orange juice is often serviceable, as is the administration daily of 1 to 2 fl. drams (3.7 to 7.4) of olive oil. In infants past the 1st year, and in older children, food may be selected of a laxative nature suitable to the age. The amount of cream used on the cereal may be advantageously increased in many instances, and the quantity of whole milk diminished. Fruit juices, and, later, fruits themselves, are excellent; especially to be mentioned here being prune juice. For older children figs and dates in moderate quantities do well if well digested. Such green vegetables as spinach, asparagus tips, and string beans, and such flours as oatmeal and Graham are of the laxative class. Bran-biscuits¹ made at home are frequently serviceable. These are quite palatable, and may be rendered still more so if broken and served with

¹ Recipe for bran-biscuit: Mix 1 pint of bran, $\frac{1}{2}$ pint of flour, and 1 level tablespoonful of baking soda. Mix $\frac{1}{2}$ pint of milk and 4 tablespoonfuls of molasses. Add this to the bran-mixture and bake in gem-pans.

milk and sugar. Increase in the amount of butter is often efficacious and a piece of butter may be given once or twice a day to a child of 2 years in addition to that used on bread and with vegetables. Broth frequently exerts a laxative action through the salts contained in it, and dextrinizing the cereal porridges with a malt-extract (see p. 155) may sometimes be done to advantage. In general those articles of diet which are largely digested and absorbed, such as the protein of milk and foods consisting chiefly of starch, should be restricted in amount, and those of fat nature or containing much waste, such as green vegetables, many fruits, and the outer coating of the grain, increased. When the constipation is simply one of the symptoms of chronic intestinal indigestion, the dietary suggested may be entirely inadmissible, and produce symptoms much more troublesome than the constipation. In such cases the food must be selected which is suitable for the digestive disturbance, and other means employed to relieve the constipation.

Training is of great importance at any age. Even when but a few months old a regular habit is favored by holding the infant in the nursery-chair at the same hours daily. Older children should be compelled to give opportunity for the bowel to empty itself at a fixed hour, at a time when the inclination is most strongly felt and when there is nothing to cause hurry. Shortly after breakfast is generally a suitable time, since the eating of food increases the intestinal peristalsis and often produces the desire for an evacuation. The disposition of children is to resist this desire unless they are instructed never to do so. Hard straining is of course not to be encouraged; but a sufficient time, 15 minutes or even a half hour, allowed in the toilet will often be followed by a stool, even though at first no inclination is felt; and a child should be taught that it can do nothing in the way of play or other amusement until the bowels have been opened.

Exercise and massage are often very valuable. Abdominal massage applied daily by one understanding it properly is of help both with infants and with older children. Not only should the abdominal muscles be kneaded to increase tone, but by a pushing movement with the flat of the hand along the course of the colon, the intestinal muscles are reached and the fecal matter aided in its course through the gut. Abundant exercise on the part of the child is a decided aid. I have seen most obstinate chronic constipation promptly relieved by the removing of the patient to a hilly summer-resort, apparently through the greatly increased amount of exercise taken.

If constipation persists in spite of a proper diet and of careful training and exercise, *medicinal measures* and other allied treatment must be employed to supplement these. They should, however, be used no oftener than necessary and discontinued as soon as possible. Suppositories are of service in this connection. These may be of soap, glycerine and soap, or gluten. Sometimes the introduction of the oiled thermometer-stem gives all the local stimulus to contraction that an infant requires. Glycerine-suppositories are too irritating for constant use. In the training of children to a daily habit suppositories may be used if no stool is obtained after a sufficient time has been passed in the chair or toilet. In a short while it is probable that this measure will be no longer required. Enemata are also useful in emptying the bowels in chronic constipation, but, like suppositories, should not be employed as a routine measure. They should be small unless there is reason to believe that there is a large fecal accumulation. Small injections of cotton-seed or

other bland oil are the least irritating and are employed to soften the feces; somewhat larger ones of well-diluted glycerin (1:10) are serviceable when there is lack of tone; and still larger ones of normal salt-solution for infants, or of soap and water at this age or in older children, where there has been no passage for several days. Enemata are less serviceable than suppositories when only local stimulation is required, as they lose their effect and need to be constantly increased in size. One of the most successful courses of treatment in chronic constipation is the giving nightly on retiring an enema of sweet oil of from 2 to 6 fl.oz. (59 to 177) or more, depending upon the age of the child and the tolerance of the bowel. In the morning the desire for an evacuation will probably be present and the softened stool passed without difficulty. In obstinate cases it may at first be necessary to administer a saline laxative in small dose before breakfast, but the need of this generally soon ceases. Stretching of the anal sphincter is occasionally very serviceable in the instances where great spasm of the muscle is present or where evacuation is evidently painful. This is, however, seldom necessary, the difficulty being overcome by other measures.

Treatment with drugs by the mouth is to be mentioned last, because, although often necessary, it is to be deprecated. In infancy some mild remedy added to the bottle-food is often of great service. Here may be mentioned manna (5 to 10 grains) (0.32 to 0.65), phosphate of soda (5 to 20 grains) (0.32 to 1.3), or milk of magnesia ($\frac{1}{2}$ to 1 fl. dram) (2 to 4). Castor oil, although laxative, commonly leaves constipation in its train, and, like calomel, should be reserved for times when distinct evidences of indigestion are also present. Some of the less bitter preparations of cascara in doses determined by trial are of great service. Senna and phenolphthalein are useful drugs, and the syrupy malt extracts are often efficacious. It is a good plan to continue no one of these substances, or indeed any other measure, without change. Thus the giving of a laxative may be replaced after a few days by the use of suppositories or by enemata. In this way the acquiring of a tolerance for the treatment is avoided as much as possible.

In subjects past the period of infancy the same internal remedies may be employed in larger amount, or such drugs used as sulphate of magnesia with syrup of rhubarb, syrup of senna with cascara, phenolphthalein in small dose, or one of the laxative mineral waters before breakfast. The same precautions should be taken as in infancy against the patient becoming accustomed to any one measure. Among some of the other remedies recommended for either infancy or children are agar-agar, exodin, purgen, purgatin and petrolatum liquidum. Agar-agar, according to Schmidt¹ contains 0.6 per cent. of cellulose and acts purely mechanically. It may be mixed with a cereal and given in the form of a porridge. Liquid paraffin (petrolatum liquidum) is one of the most popular. This, too, acts mechanically, and may be administered plain or made into an emulsion with an aromatic water. Children old enough to take pills may receive small doses of aloin or of podophyllin. At all ages drugs which increase the tone of the intestinal or abdominal muscles are useful, especially to be mentioned being *nux vomica*.

¹ Münch. med. Wochenschr., 1905, 1970, Oct. 10.

CHRONIC INTESTINAL INDIGESTION

This exceedingly common disorder affecting both infants and older children is very frequently associated with gastric indigestion, but probably still oftener occurs alone. It is, strictly speaking, a purely functional disturbance, yet in symptoms it may sometimes not be readily distinguishable from mild cases of ileocolitis characterized by actual inflammatory lesions. It is one of the most difficult diseases to treat, especially in infancy.

A. CHRONIC INTESTINAL INDIGESTION IN INFANTS

(Intestinal Dyspepsia; Decomposition)

Etiology and Pathology.—The disease is much oftener seen in the 1st year of life and especially in the first 6 months. Poor hygienic surroundings predispose, as does very greatly a congenital constitutional debility. This last is a common factor, the infants in many cases having been born prematurely or being the offspring of tuberculous or syphilitic parents, or of others with some unfavorable constitutional influence in the way of parentage. Among frequent causes is the occurrence of attacks of acute intestinal indigestion or acute ileocolitis. Of all etiological factors, however, the continued employment of an unsuitable diet is by far the most influential and common. It is on this account that the disease is much oftener seen in those artificially fed, although it is true that breast-fed infants may readily develop it if the breast-milk is of unsuitable character.

In *breast-fed children* the fault is sometimes easily ascertained. The quantity of milk secreted by the mother and taken by the child may be constantly too great. In other instances the supply is far from sufficient and the infant's general health consequently suffers, until finally the intestinal functions are so weakened that an amount of food which will be properly nourishing cannot be assimilated. In still others—and these the most frequent of all—some of the constituents of the breast-milk are easily discovered to be secreted in excess. Many times, however, analysis reveals nothing whatever which can account for the persistent indigestion present. Sometimes it appears to be some constitutional trouble with the nursing mother, as when, for instance, she is of a highly neurotic temperament, or shows other evidences of ill health, although the way in which the milk is affected is not discoverable.

In the case of *artificially fed infants* the fact that the diet is necessarily an unnatural one renders it unfit in any form for many infants, and chronic indigestion is an unavoidable result. The chemical differences in the proteins and fats make it impossible to prepare a milk modification exactly like human milk, and in addition is the lack of similarity in the ferments, salts and other bodies, the importance of the action of which is still little understood. Oftener there is a very evident and clearly recognizable fault with some one of the ingredients, an amount of this being given which the digestion cannot tolerate. Besides this comes prominently into play in bottle-fed babies the element of infection of the milk by germs of various sorts. (See *Acute Gastroenteric Intoxication*, p. 738.) Finally it frequently happens in well-advanced cases that although a change in an unsuitable milk mixture would have been efficacious if made earlier, the infant when coming under observation has reached a condition where no alterations of the diet attempted have any

beneficial effect, the child being unable to make use of the elements supplied.

Just what the principal defects in the diet may be in cases in bottled infants is a matter much discussed and far from ultimately settled. This subject has already been considered to some extent elsewhere. (See *Action of the Different Food elements in Digestion*, pp. 99, 127, 178; *Finkelstein's Classification*, p. 697; *Feces in Disease*, p. 731.) Undoubtedly too much blame was formerly placed upon an excess of protein, and especially of casein; and the disposition now is in many quarters to attribute indigestion solely to the whey, the carbohydrates, or the fat. Physicians who have repeatedly seen good results from the use of whey hesitate to assign to it all the evil influences claimed by some observers. Doubtless there is some middle ground of belief which will eventually be established. There is at any rate a very general agreement at the present time that the fat of the milk is one of the ingredients most difficult to digest. Less often the sugar causes trouble, but to what extent this is an independent action, or how much it acts synergetically with other elements of the food is still uncertain. The addition of amylaceous food to the nourishment aids digestion in many cases; and in others is certainly the cause of chronic intestinal indigestion. Many of the proprietary infant's foods probably act harmfully through the high percentage of unconverted starch present.

The whole matter is still in the process of solution. It must be recognized, however, that, although there may be a general rule evolved, and although fat indigestion is certainly a very common form, yet the question is, to some extent, an individual one and has to be determined largely for each child.

Here may be again mentioned the fact, emphasized by many writers, that the trouble appears not always to be in the intestine itself, although perhaps primarily so, but in the defective metabolism in the tissues of the body. This is particularly true of the advanced cases where the condition of infantile atrophy (p. 610) finally develops. It is not only the failure to absorb food to a sufficient extent which is at work in these cases, but the harmful action of abnormal substances produced in the intestines or in the tissues.

Pathological Anatomy.—In typical cases there are no lesions found, the disease being a purely functional disturbance. In advanced cases, however, complicating secondary lesions appear, among them inflammation of the intestinal mucous membrane. The disorder then has changed to a condition of chronic ileocolitis.

Symptoms.—In some cases there is constant diarrhea, the stools being watery, greenish, and containing curdy masses of various sizes. The number is seldom large, they are passed without pain, and mucus is seen in small amount only, if at all. If later in the disease there is constantly a large amount of mucus present, it is probable that a chronic ileocolitis of mild grade has developed. In other cases the stools are only occasionally diarrheal, or there may be a chronic constipation, the passages often being pasty and too light in color and sometimes hard, either in small scybalous masses, or in larger form with difficulty in evacuation and requiring enemata or purgatives. In any event microscopical and chemical examination often reveals undigested food, especially fat; free, or in the form of soap. The odor of the stool, whether loose or formed, is generally unpleasant or sour; and occasionally offensive if there is decomposition of proteid material going on. Vomiting occurs occasionally in

most instances, but is not a constant or troublesome symptom unless the disease is complicated by gastric indigestion. The abdomen is usually distended by gas and there is frequent colic if constipation is present, but less often so if diarrhea. The appetite is generally good and sometimes large; the tongue varies; there is often irregular fever alternating with normal or low temperature, or there may be more constant elevation, but only when symptoms of constitutional intoxication develop. The urine may show the presence of the acetone bodies and an increased output of nitrogen in the form of ammonia, but this is not always the case. In some patients there arises an intolerance for cow's milk in any form, its administration being followed by an exacerbation of the symptoms, including vomiting, diarrhea, fever, and sometimes cutaneous eruptions and evidence of a disturbed nervous state.

The chief symptom, however, is *persistent increasing malnutrition*, with all the symptoms already described under the heading of infantile



FIG. 252.—CHRONIC INTESTINAL INDIGESTION.

Child of 3½ months, in the Children's Hospital of Philadelphia. Great wasting; moderate fever; bowels loose; failure to improve under any treatment; death.

atrophy (pp. 612 and 698). The children gradually waste more and more, and suffer from low temperature, feeble circulation, anemia, and increasing debility. They are usually constantly fretful in the early stage and often apathetic later. During this condition of malnutrition no very positive evidences of indigestion may be discoverable. Yet diminishing the amount of food of these infants may increase the rapid loss of weight, while increasing the amount may have the same effect, often with attacks of autointoxication of a dangerous nature.

Course and Prognosis.—The course of the disease is very variable and the duration uncertain. At best it is long-continued and lasts for months before recovery is assured. In some instances the loss of weight is constant and extreme (Fig. 252). In others there may be long periods during which it is stationary or even in which temporary improvement and increase of weight occur, to be followed, however, by relapse. In still others, not too far advanced when coming under observation, there is eventually a more or less steady increase of weight and return to general health. In nearly all, however, there are liable to be exacerbations, depending probably upon intercurrent acute dyspeptic disturbances of

the stomach and intestines perhaps advancing to the stage of intestinal toxemia. Vomiting may then for a time become troublesome or diarrhea be a marked symptom, while fever develops, and the nervous symptoms characteristic of the condition (pp. 698 and 741) may appear (Fig. 253).

The *prognosis* is always serious, especially in cases with diarrhea, and in those others in which, with little active intestinal disturbance, improvement in weight fails to take place no matter what change in diet is made. In the majority of such cases death follows finally from exhaus-

tion or intoxication, perhaps during an exacerbation.

The earlier in the disease the treatment is commenced the greater is the chance of recovery. On the other hand, an infant who has long tolerated the disorder may be assumed to possess greater resisting power. Serious although the disease is, even apparently hopeless cases sometimes recover under proper treatment, but without this there is little chance for improvement. There is, moreover, constant danger of relapses from slight or undiscoverable causes. When recovery does take place it is usually finally complete and the condition of the child in the 2d or 3d year seems often to be no worse as a result of the illness in the 1st year of life. Some patients, however, continue delicate, or later suffer more or less from intestinal disturbances.

Complications.—These are numerous and are largely those described under ileocolitis and infantile atrophy; consisting of thrush, erythema and intertrigo especially of the anal region if there is diarrhea, furunculosis or other suppurative processes of the surface of the body, suppurating otitis, and atelectasis. There may be widespread petechiæ before death, and often terminal pneumonia,

nephritis, or convulsions may develop. The disease may readily pass into an ileocolitis of a chronic nature and result fatally.

Diagnosis.—The advanced cases are to be distinguished chiefly from *infantile atrophy* depending upon other causes, such as congenital asthenia, tuberculosis, and syphilis. The history of early digestive troubles preceding the atrophy is generally sufficient to confirm the diagnosis of chronic intestinal indigestion, if no positive symptoms of constitutional disorders contraindicate this. The diagnosis is also to be made from milder, long-continued cases of *chronic ileocolitis*. Both diseases

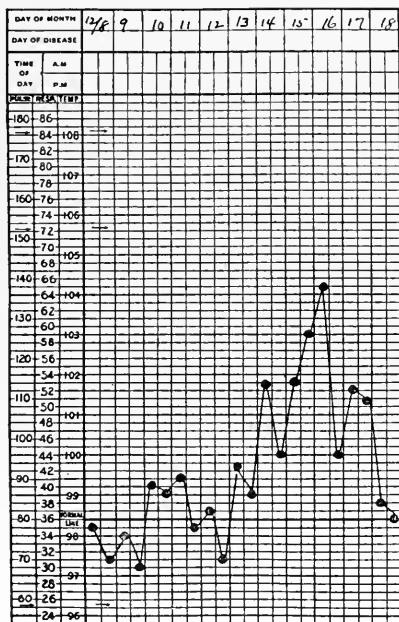


FIG. 253.—EXACERBATION OF SYMPTOMS IN A CASE OF CHRONIC INTESTINAL INDIGESTION.

George B., aged 6 months. In the Children's Hospital of Philadelphia for chronic intestinal indigestion. Occasional vomiting; 2 to 3 slightly undigested and diarrheal stools daily; much emaciated. Dec. 12, suddenly developed apathy; toxemic symptoms; prostration; distended abdomen; 5 to 7 diarrheal stools daily, greenish, with pus and mucus; increase of temperature. Weight 7 pounds, 7 ounces. (3374.) Final recovery.

produce wasting and exhibit disturbances of the stools; but in indigestion these stools fail to show the constant large amount of mucus which is diagnostic of chronic ileocolitis. As already stated, the passing of the functional into the inflammatory disorder is of great frequency. The recognition of the cause of the chronic intestinal indigestion is important but often very difficult. A careful consideration of the earlier history of the case, with special reference to the nature of the diet employed, is frequently a great aid in this direction. An excess of carbohydrate in the food produces colic and very possibly watery, irritating, acid movements, often with an penetrating acid odor suggesting acetic acid; sometimes offensive, if there is a combined protein-indigestion. Excess of protein may occasion either constipated or diarrheal stools with a putrefying odor, and protein-curdls may be present. Excess of fat may also result in either constipation or diarrhea. In the former the stools are of a grey or white color, are composed largely of insoluble fatty soaps, and may have an unpleasant smell. When diarrhea is the symptom the stools may be pale-yellow, with a sour or offensive odor, more or less glistening from the presence of fat, and reveal under the microscope fat-globules and crystals in excess. In many other cases they are watery, mucous, greenish, and with numerous curdy masses of a whitish color. (See *Feces in Digestive Diseases*, p. 731.)

Treatment.—*Medicinal* treatment is purely symptomatic. Constipation is to be relieved by enemata or gentle laxatives; occasionally by free purgation if there are symptoms of acute gastric or intestinal indigestion. Diarrhea may be checked by appropriate means. (See *Acute Intestinal Indigestion*, p. 736.) Colic is to be treated by measures already referred to for this disorder (p. 730). The strength must be sustained by stimulants, the body temperature by external heat. During exacerbations brief starvation must be instituted and a purgative given. This is especially true if symptoms of intoxication have appeared.

In the line of *hygienic* treatment, care is required that the infant have plenty of fresh air. This is a matter of great importance. It is well recognized that infants with malnutrition, confined to close hospital wards, do not thrive, whether chronic intestinal indigestion or other disorders be operative. The infant may be kept in bed and the windows of the room open; or if well enough be taken out of doors daily, the bodily temperature in cool weather being maintained by the free use of hot-water bottles. It is only when the infant's temperature is decidedly subnormal, or when exposure to cooler air is followed by a dangerous depression of the child's temperature in spite of efforts to prevent this, that the airing described must be avoided. In such cases the infant must perforce be kept in a well-warmed room until sufficiently improved to bear the cooler air. In very feeble infants rubbing the body under the covers with warm oil should replace the bath. In others a daily sponge-bath or tub-bath is of advantage, provided a good reaction follows.

By far the most important treatment, however, is *dietetic*, and this is frequently one of the most difficult problems which the physician ever encounters. In the cases of chronic indigestion in *breast-fed infants* too great haste must be avoided in advising weaning. I cannot too strongly insist upon this point. Sometimes a regulation of the mother's diet and method of life, or some alteration in the frequency of feeding the infant will suffice. In other cases, the employment once or twice daily of an artificial food may answer, breast-milk being still the principal diet; since the giving of even a small amount of human milk seems frequently in

some way to enable the infant to digest more satisfactorily a diet largely artificial. Some infants in spite of evident failure to digest breast-milk perfectly continue to thrive in other respects, and in such it is certainly well to delay the withdrawing of breast-feeding, inasmuch as there is no surety that artificial feeding may not have a worse result. The temporary withdrawal of the breast for 24 or more hours at a time may suffice in such cases. When, however, the indigestion is persistent and the child is losing ground in spite of faithful efforts to remove the difficulty, either the employment of a wet-nurse or the institution of weaning becomes necessary.

In the dietetic treatment of *artificially fed* infants with chronic intestinal indigestion, the first thing required is a very careful survey of the previous history in the effort to determine what element of the food, or fault in the preparation, has originally produced the disease, or has maintained it. The minutest details should be ascertained, and long-continued, persistent study may be necessary. Not only the character of the food must be considered, and the degree of digestion of this, especially by an examination of the stools, but the quantity given, the intervals of feeding, the rapidity of taking nourishment, the amount of dilution or concentration, and many other circumstances. If active symptoms are present, dietetic treatment may well begin with a moderate starvation for 24 hours or longer. For this purpose some thin cereal decoction such as barley water or arrowroot water, is generally of service. After this, some form of diet of weak strength must be selected until better digestive conditions are established, and a more permanent food-supply of a satisfactory character can be tolerated. Here there is a large choice, depending upon what the dietetic cause and the character of the symptoms appear to have been. If the study of the stools (p. 731) shows that the fat is especially difficult of digestion, as is true in the large majority of cases, milk-mixtures made from skimmed milk are suitable, or, still better, in my experience, those from buttermilk. The degree of dilution will depend upon the tolerance for protein. Inasmuch as the caloric value of these foods is low, they may often be strengthened by the addition of carbohydrate in some form. If there is difficulty in the digestion of sugar, as shown, for instance, by acid diarrhea, with irritation of the buttocks, buttermilk is of especial value on account of its low sugar-content (p. 147). Casein-milk in some of its forms is serviceable for the same reason, having a low percentage of carbohydrate present, unless sugar has been added to it (p. 148). Since there is a difference in the digestibility of the sugars, it is often possible to fortify the food by the addition of saccharose; or of a dextrine-maltose preparation (p. 129) in place of milk-sugar. Often, however, maltose-preparations will increase the frequency of the diarrhea. It is well also to increase the alkalinity of the maltose-mixture by the addition of bicarbonate of potash.

In very many conditions the addition of an insoluble carbohydrate such as a thin or thick cereal decoction is of great advantage, its strength varying with the case. The mode of action of this ingredient is uncertain. The starch is to an extent digested and utilized, and to an extent also acts mechanically as a protective colloidal substance. Certainly many infants do better when the plain water used as a diluent in the mixture is substituted by a cereal water. The employment of starch serves a purpose not attained by the addition of soluble carbohydrate. It is for this reason that the use of the various malt-soups is often more efficacious than the addition of sugar or of dextrine-maltose preparations

alone. (See Malt-soup, p. 156.) The casein of the food is less frequently a source of indigestion, but undoubtedly can produce it, especially in the early months of life. The symptoms are indefinite, but often consist in flatulence, colic, constipation, and an offensive odor to the stools; less often in the passing of casein-curd (p. 131). Under such conditions, whey is frequently a valuable remedy. Its caloric value is low; but in spite of this young infants will frequently thrive on it for a time. Its nutritional value may be increased by the addition of small amounts of strong cream, when no fat-indigestion is present. The amount of casein added in this way is inconsiderable. In some cases where fat, too, is not tolerated, the whey should be made of skimmed milk, and white of egg may be added to it, and sugar also, preferably dextrine-maltose preparations, if found desirable. The employment of a malt-soup is sometimes useful here, inasmuch as it contains both soluble and insoluble carbohydrate. In still other cases the digestibility of the casein of the food may be increased by peptonizing, but this is not so often needed as formerly believed.

In many instances an intolerance for any form of milk develops, usually, however, only temporary, and some other kind of nourishment must be employed for a time. Here the cereal gruels and albumen water are often serviceable. The gruel must, however, be sufficiently strong to give it some real food-value.

As regards all these methods described, it is to be remembered that, as a rule, they are to be looked upon as temporary procedures, to be carefully changed when the digestive disorder has been abated. It is equally necessary, too, to make our first effort the allaying of the symptoms; not the increasing of the weight of the child. So long as the strength is maintained, and the weight remains stationary or diminishes but little, the effort to cause a gain in this respect should be deferred until the other symptoms are sufficiently relieved.

Besides the ingredients of the food, its dilution and its frequency of administration must be considered. Some infants do better on a diluted food; others upon smaller amounts of a more concentrated nourishment. Only trial can show which is to be preferred. As a rule, too, it is better to make the intervals of feeding decidedly longer than in health. A 3-hour or 4-hour interval in the early months of life may be better than one of 2 or 2½ hours. Still another factor is of importance, namely the mutual influence which the different food-ingredients exercise upon each other. For instance, the addition of a high carbohydrate-percentage may make both the protein and the fat more digestible, as seen, for instance, when malt-soup is employed; or fat may be tolerated if the whey is diminished in amount. (See pp. 49, 50, 129, 130.)

The method of changing from any one of these substitutions mentioned to one containing the usual elements in more normal ratio is often a difficult matter which must be determined for the individual case. Thus an infant taking whey may have this fortified by egg-albumen, and later, in cases of casein-indigestion, after the symptoms have been relieved, by the gradual addition of peptonized milk; or if fat-indigestion has been present also, first of skimmed milk and then of whole milk. A baby with sugar-intolerance fed upon casein-milk may gradually have increased amounts of saccharose or maltose added. One with fat-indigestion, fed upon buttermilk, may after a time have small amounts of cream added. Certain practical points in the method of preparation must be borne in mind in making any such changes. Thus, before mixing

cream or milk with whey, the latter must be heated sufficiently to destroy the rennet, or the casein will be coagulated. So, too, in fortifying whey with cream, the fat-percentage of the latter must be considered in order to obtain the result desired. It will be readily seen, for instance, that using a cream of, say, 30 per cent. fat-strength, the addition of 1 part of this in 30 parts of the food increases the fat by 1 per cent., but raises the percentage of casein to so small an extent that this may be ignored entirely. Were a weaker cream used, much more of it would be required, and the casein might be increased undesirably. Milk or cream added to buttermilk will be coagulated by the acid present, and the mixture must be shaken well and not allowed to stand any length of time before it is given.

With the use of the substitutes for milk-feeding in the form of the very numerous proprietary foods on the market, I have had a large experience in cases seen in the practice of other physicians. My belief is that little if anything is to be gained by their employment which cannot be obtained by some of the methods already outlined, and the lack of sufficient general knowledge of their composition prevents physicians, as a rule, from using them intelligently. In no disease is the study of the individual of greater necessity than in chronic intestinal indigestion, and this is seldom made when proprietary foods are ordered. It is only by this study and by the consideration of the apparently unimportant matters that success can be obtained, whatever the food employed.

Many cases are encountered, however, in which no change of diet made appears to influence in the slightest the general condition of the child. There may be neither vomiting nor diarrhea, but the weight remains stationary or gradually diminishes. The infant has, in fact, reached the condition described by Finkelstein as "decomposition" (p. 698), where it is no longer able to benefit by any artificial food given. In such cases nothing remains but to obtain a wet-nurse if possible. This often avails in a surprising manner; but often, too, fails, because it has been deferred too long, and the digestive functions have finally become unable to utilize even the infant's natural food. To wait for such a condition to develop before advising wet-nursing is bad management on the part of the physician. On the other hand, the procuring of a wet-nurse, valuable as this so often is, can by no means be regarded as a certain cure for intestinal indigestion; even before the serious condition mentioned has been reached. In many instances, particularly where there is much fat-indigestion on the part of the intestine, or where vomiting shows the presence of a complicating gastric indigestion, undiluted average human milk is too rich for the earlier part of the treatment, although it may be the best food later.

In this connection reference must be made to the attempted treatment of intestinal indigestion and of diarrhea by the direct modification of the bacterial flora of the intestine. As pointed out by Kendall and Smith¹ and others, there is a group of bacilli in the intestine claimed to be causative of the symptoms seen, the multiplication of which can be checked by either the direct administration of other germs which interfere with their growth, or by the giving of food which contains or favors the increase of the bacteria desired. The Bulgarian bacillus and other lactic acid organisms are especially to be mentioned here, their presence inhibiting the development of the dangerous proteolytic bacteria. Consequently, the employment of buttermilk which has not been heated

¹ Boston Med. and Surg. Jour., 1911, CLVII, 306.

after it was made, or of other milk containing the organisms mentioned, or the direct administration of cultures of these germs, is curative to a disturbed intestinal digestion in a number of cases. This view has found acceptance in many quarters, and good results have repeatedly been reported. It is impossible as yet to determine the true value of the method, or to know to what degree the supposedly harmful germs are directly injurious, or to what degree they are a secondary accompaniment of the disorder.

B. CHRONIC INTESTINAL INDIGESTION IN OLDER CHILDREN

This exceedingly common affection in children past the period of infancy may manifest itself in typical form, or may give rise to symptoms which are very confusing. Disturbance of the stomach may be combined with it.

Etiology.—A very frequent cause is persistence with the ingestion of carbohydrates in large amount. Any starchy food may give trouble, but potato is one of those most liable to do so. In other cases too long a continuance of milk as the chief article of diet may bring it about. The giving of candies and other sweets; allowing the child to eat when it pleases between meals; in some cases an excess of fat in the food; and swallowing without sufficient mastication are among other dietetic influences. Nourishment of any sort which is poorly prepared or of an indigestible nature is likewise a cause. The disease is consequently common in children who receive food from the family table at too early an age. An alteration of the bacterial flora of the intestine was emphasized by Herter¹ (see p. 533) as the cause in a certain class of cases; the bacillus bifidus, bacillus infantilis, and coecal forms characteristic of infancy persisting, and the bacillus coli and bacillus lactis aerogenes, which should normally be present at this age, being absent from the feces. Apart from this, any debilitated condition of health predisposes. Consequently the disease frequently follows some acute diarrheal disturbance, or is associated with rachitis, or develops after some exhausting, acute disorder, and is one of the conditions often occurring in neurotic children.

The majority of cases come under observation between the ages of 3 and 10 years, although the disease has not uncommonly begun in the 2d year of life. The social position is without influence.

Pathological Anatomy.—As in infancy, the disease in a strict sense is a functional disturbance, no lesions of the intestine being present, or being limited perhaps to injection and redness of the mucous membrane and an increased secretion of mucus. Both the small and large intestine are commonly much dilated.

Symptoms.—In well-developed typical cases, the symptoms are very characteristic. They are those rather of chronic toxemia than of local intestinal disturbance. There is a very decided loss of flesh, the limbs especially being thin and the child having a delicate appearance, with an anemic or sallow complexion, dark rings or puffiness under the eyes, and perhaps a slightly yellow tint to the sclerae. The pallor is sometimes replaced for a time by a red flush of the cheeks, and on other occasions shows a great temporary increase, as though the child were faint or nauseated. The appetite is variable and capricious and generally very poor, although in some instances excessive. Eructation of gas is com-

¹ On Infantilism from Chronic Intestinal Infection, 1908.

mon, as is its passage from the bowel, and the abdomen is usually much distended and tympanitic. This abdominal distention is one of the most characteristic symptoms. Nausea and vomiting may occasionally occur if the stomach shares in the dyspeptic condition, but in some instances vomiting and headache appear to depend upon intestinal toxemia. The tongue is pale, flabby and perhaps tooth-marked; sometimes coated; sometimes exhibiting enlarged papilla. It has seemed to me that the geographical tongue (p. 667) is particularly liable to be found in this disease, although of course not confined to it. The odor of the breath may be offensive, but this and the condition of the mouth and tongue depend largely upon the gastric disturbances frequently associated. The bowels are usually constipated, or constipation may alternate with attacks of diarrhea. The color of the stools is generally pale and sometimes nearly white; at other times brownish. They are frequently offensive in odor, contain undigested food, and when loose are often frothy in appearance. Mucus is passed at times, perhaps in large amount. The mucus is mixed with the stool when this is loose, or coats it when formed. The element of abdominal pain is very variable, being somewhat colicky and paroxysmal, and frequently accompanying an evacuation of mucus. Oftenest it is slight, and sometimes is no more than a sensation of abdominal discomfort. The urine is not characteristic. At times it may show the presence of indican in considerable amount, or of other bodies denoting indigestion. The general health suffers and the children develop poorly in height and weight.

The nervous symptoms are many and varied. In fact they are often the most prominent manifestations of the disease, and may readily lead to a mistaken diagnosis. Although the child is mentally unaffected and often very bright and even precocious, he is irritable, hypochondriacal, languid, and easily tired. The hands and feet are cold, and the skin perspires readily. Sleep is nearly always restless and tossing, with frequent grinding of the teeth. Outcries and dreaming, and not infrequently night-terrors and somnambulism are observed. Wakefulness is not an uncommon symptom. In some cases there is stupor, or even tetany or convulsions. Shortness of breath is sometimes witnessed, or the respiration may occasionally be sighing in character. In other instances asthmatic symptoms of digestive origin may be present. There is usually little or no fever, or perhaps the constant presence of an elevation of temperature of less than, or even slightly over, 100°F. (37.8°C.); except during the occurrence of exacerbations when the temperature rises considerably. The pallor of the skin does not necessarily depend upon an actual anemia. In some cases the skin is unnaturally dry; in others there is an urticarial or erythematous eruption.

The group of symptoms as described is not seen in its entirety in every instance. In the milder cases, or those which have lasted but a short time, the disease is rather a series of acute attacks of moderate severity, with intervals of comparative health, and with but little disturbance of the general condition. In others symptoms of a severe type, with a degree of wasting suggestive of pulmonary tuberculosis, are attended at times by the passing from the bowel of unusually large amounts of mucus. To this complex of symptoms the title "Mucous Disease" was applied by Eustace Smith.¹ Another form of the affection was called "Celiac Disease" by Gee.² Here the predominant symptoms are

¹ "Wasting Diseases of Infants and Children," 2d Amer. Ed., 156.

² St. Barthol. Hosp. Rep., 1888, XXIV, 17.

the pasty, white, offensive, and poorly digested stools, containing an excess of fat and moderately increased in frequency and especially in size; wasting; debility; and often retardation of growth. Well-marked cases of this type have been denominated by Herter¹ "Intestinal infantilism." (See Vol. II, p. 533.) These always exhibit evidences of unusual putrefaction of the intestinal contents and the presence of large numbers of the bacillus bifidus and the bacillus infantilis in the feces, instead of the usual bacillus coli. There is not only a decomposition of protein, but a failure to absorb the carbohydrate and fat, together with a loss of calcium and magnesium in the form of soaps.

Course and Prognosis.—Children with chronic intestinal indigestion are usually slow in recovery, even under treatment, the disease often lasting for years. There is a tendency to acute exacerbations of gastric or intestinal disturbance. These occur at irregular and often frequent intervals, are provoked by slight and often undiscoverable causes, and are characterized by nausea, vomiting, diarrhea, slight fever, increased loss of appetite and a greater manifestation of nervous symptoms. In the intervals between the attacks the evidences of indigestion may at first be very slight, or in the mildest cases wholly absent; but it is not long before there is a fuller development and increase in the severity of the symptoms until some signs of the disorder are constantly present. The course of the disease at best is chronic, its length depending upon the nature of the cause, the severity of the symptoms, the duration before the treatment was commenced, and the patience and faithfulness with which this is carried out by the parents. Only in mild cases, or in those severer ones in which the errors in diet and hygiene have been very evident and consequently easily corrected, can we hope for more rapid improvement. In the milder cases there is, it is true, a certain degree of natural tendency to recover when puberty is reached; but this can in no way be depended upon, and, as a rule, there is little likelihood of spontaneous cessation in untreated cases. The prognosis of those under treatment is on the whole good, except in the instances where little can be found wrong with the hygiene and diet, and where there seems to be a constitutional, or an early acquired and finally firmly seated, lack of functional intestinal power. There is always, too, the danger in this disease that the impaired general health may diminish the power of resistance to some intercurrent affection such as bronchopneumonia.

Diagnosis.—The tympanitic condition in intestinal indigestion may be so great that I have known cases erroneously diagnosed as *idiopathic dilatation of the colon*, and have been able to prevent dangerous and unnecessary operative interference. The two conditions are quite distinct in their history and symptoms. The nervous symptoms may be confusing if the condition of the digestion is not carefully studied, but with this exception the diagnosis is usually not difficult. The slight cough which may accompany the wasting can suggest pulmonary *tuberculosis*, but examination of the lungs fails to reveal any anomaly. A negative von Pirquet reaction would be of value. Tuberculous peritonitis exhibits tenderness and either fluid or some evidence of deposit in or thickening of the abdominal walls. The presence of *intestinal worms* may produce grinding of the teeth, abdominal pain, disturbed sleep and other nervous symptoms; but the administration of a vermifuge will reveal the cause and clear up the diagnosis. Convulsions in childhood are often considered to be a manifestation of *epilepsy*, when they really

¹ *Loc. cit.*

depend solely upon chronic intestinal indigestion. To complete the diagnosis of indigestion a careful and even a microscopical examination of the stools may be required, demonstrating that the food is passing in an undigested form and showing the exact nature of this.

Treatment.—*Dietetic* treatment occupies the primary place. First in this connection is to be considered the manner of eating. Attention must be paid to the condition of the teeth; since carious teeth, especially if painful, may render proper mastication impossible. Apart from this, there is a natural disposition in early childhood to eat with little mastication, and this readily becomes a fixed habit unless carefully guarded against. The life must be so ordered that there may be sufficient time to eat slowly. Partaking of a hurried breakfast in order not to be late for school is a common cause of chronic indigestion. The haste of the luncheon at school is often as harmful as the improper nature of the articles frequently eaten at that time. Not too large an amount of food must be ingested at any meal. On the other hand, since the appetite is often poor, meals may need to be more frequent than in health in order to obtain the taking of a sufficient amount of nourishment. Meals should be at regular intervals and no food at all given between them.

Those articles must be excluded from the dietary which are most liable to cause indigestion, or which have been found to do so in the individual case. This individuality is an important matter; what agrees with one child perhaps disagreeing with the next. In general, however, as already stated, the food most frequently the cause of the symptoms consists of the carbohydrates and especially the starches, and next to these in importance fat in any form. Consequently, in bad cases it would be best to eliminate starch and fat almost entirely for a time, until improvement is well established, the child eating only meat, skimmed milk, buttermilk, beef-juice, broths, oysters, white of egg, green vegetables and similar substances. Such vegetables may be chosen as lettuce, spinach, asparagus, vegetable-marrow, stewed celery, and tomatoes, the carbohydrate content of which runs from 2 to 3 per cent. (Joslin).¹ As it is difficult to institute such a restricted diet in children, and as the milder cases do not require it, we may generally obtain good results by merely limiting the amount of starch very greatly. Potatoes should not be given at all, since their digestibility is certainly less than that of some other amylaceous foods. As little bread should be used as possible. It should be stale, thoroughly toasted, or in the form of zwieback, only a small piece being given with the meal. Some unsweetened commercial biscuit, such as water crackers, oyster crackers, and the like, taken in small amount, may replace bread at times. Among other foods which must be used with caution on account of their carbohydrate content, are white and lima beans, peas, carrots, corn, beets and parsnips. Cereal porridges must likewise be partaken of in very small amounts, if at all, and such as oat-meal or those of whole wheat avoided, on account of their more irritating character. Arrowroot, rice and farina are to be preferred. Even the soluble carbohydrates, the various sugars, maintain the state of indigestion. This is particularly true of candies, jellies, jams, and preserved fruit. Consequently, these are to be avoided entirely, or very small amounts of cane-sugar employed as a necessary sweetening, and this only if the child will not do without it. Sometimes saccharin may be substituted for sweetening purposes. Most fruits come in the same category as sugars, and should not be given until improvement

¹ The Treatment of Diabetes, 1916, 401.

is distinctly under way. Baked apples, prune-juice and orange-juice are, perhaps, the best. What is said of other sugar-containing foods is true also of the various dextrinized breakfast cereals on the market. Their administration should be deferred at the beginning, and then commenced as an intermediate step in the return to ordinary cereal foods.

Another very powerful factor in producing indigestion is fat, and in some instances it is this, rather than the starch, which is causing trouble. Consequently, all fats, including butter and the yolk of egg, must be prohibited in such cases, the fat removed from any meat given, and skimmed milk or buttermilk used in place of whole milk. All fried food and pastry are to be rigorously excluded. Cocoa is sometimes very well tolerated; sometimes not.

A diet rich in protein is often one of the best. Broiled or roast beef, mutton, or chicken, is usually well borne, in amount depending upon the age of the child. For young children it should be scraped or minced finely, and this may be necessary for older ones as well, if thorough mastication cannot be successfully insisted upon. Beef-juice is useful, although not very nourishing unless in larger amount than can be given with success in most cases. Broth with the meat-fibre retained in it in finely divided form is serviceable, but thickening with any starchy addition must be avoided early in the case. The fat should be thoroughly removed from the broth before this is given. The best of the proteid foods in most of these cases undoubtedly is milk free from fat, and this should form a large part of the diet. If there is difficulty in digesting the casein, the milk should be partially peptonized; or even completely so, if the child can be induced to take it in this way. Buttermilk is sometimes very serviceable. Eggs form a valuable food for many children, but are not well borne by many others. It is rarely advisable to allow them every day, and at first only the white of the egg should be given. Boiled or baked fish is often useful, and oysters, raw or very slightly stewed, are very well digested by some patients. Sometimes one of the high-protein proprietary foods on the market may be employed. (See p. 165.)

Useful vegetables in many instances are string beans mashed through a colander, spinach, squash, stewed celery, stewed salsify, asparagus tips and lettuce. The time of beginning these varies with the age of the child and with the necessity of finding some food to take the place of the articles which are forbidden. When possible, their use should be deferred for some months in cases at all severe. Young cauliflower, Brussel sprouts and onions are theoretically valuable members of this class of vegetables, but their strong odor and taste often make them undesirable.

Last of all, when other foods are found to be well tolerated and convalescence is well established, but even then only after a period of some months, a very careful return to the ordinary use of starch may be attempted, allowing only small quantities at first. It may be that potato cannot be given at all for a long time. Months, or a year or more, may be required to effect a cure. In the event of a relapse occurring, the diet should at once be reduced greatly, employing the simple regimen with which the treatment was commenced. An absolute skimmed-milk diet for a short time is often successful on these occasions.

A diet-list for milder cases of chronic intestinal indigestion, in which a certain amount of starch is permitted and in which whole milk and eggs are tolerated, would read somewhat as follows, varying, of course, with the age and the individual requirements:

Breakfast—7 A.M.: Milk with lime water; soft boiled egg or mutton-chop, fish or cold beef; a slice of stale bread without butter, or zwieback, water-crackers or similar unsweetened biscuit.

Lunch—11 A.M.: Milk, or broth free from fat.

Dinner—2 P.M.: Roast or broiled chicken, beef or mutton free from fat, or sweetbread; spinach, string beans, stewed celery, stewed salsify or asparagus tips; bread or crackers as at breakfast. For dessert junket, baked apple, or a gelatin food.

Supper—6 to 7 P.M.: Milk, buttermilk, or broth; with bread as at breakfast.

Hygienic treatment is also of importance. A daily cool morning sponge is conducive to improvement of the general health. There must be sufficient sleep in a well ventilated, cool room. The mid-morning nap should be continued as long as the child can be induced to take it. If this period is past, at least an hour's rest, recumbent, in the middle of the day, preferably before the mid-day meal, is very useful in many cases. Life out of doors is important. Whether or not the child shall attend school must be determined for each case individually. Against the confinement in school is to be balanced the disadvantage of lack of occupation and of companionship which staying at home often entails. Fatigue, either mental or physical, is to be carefully avoided. Clothing must be regulated to provide sufficient warmth without occasioning too free perspiration on exercise. Bare legs are not to be allowed, since the chilling which is liable to occur predisposes to interference with digestion. Massage is serviceable for patients too debilitated to take sufficient exercise. Change of climate is often most beneficial.

Medicinal treatment is largely a secondary consideration. The overcoming of the constipation, which is such a common symptom, is important. In bad cases the nightly injection of cotton-seed oil may be of service. (See Constipation, p. 757.) Very frequently the diet may be successfully selected to overcome the constipation, bearing in mind, however, that many of the laxative foods are more or less irritating to the bowels. As a rule enemata are not efficient, as they operate only upon the rectum, and a drug is required which affects the whole intestinal tract. In addition to such laxatives as cascara, senna, phenolphthalein, and the like, it is well to obtain a thorough purging at intervals of from 5 to 7 days, employing calomel or citrate of magnesia for this purpose. Where there is much secretion of mucus from the bowel, saline douching is sometimes of service, but this practice should not be continuous as it sometimes maintains the symptom which it is intended to cure. The administration of a good malt-extract appears to aid the digestion of starch. One should be selected known to contain a large percentage of diastase, there being a great difference among them in this respect.

In general, so far as the administration of drugs goes, I have had the best results with the combination of an alkali with a bitter tonic. For this purpose, bicarbonate of soda may be given with tincture of nuxvomica, compound tincture of gentian and an aromatic water, administered 10 to 20 minutes before meals. In other cases, a small amount of sulphate of magnesia, cascara, or syrup of rhubarb may be added, or a laxative mineral water employed, if constipation is a troublesome symptom. Cod-liver oil, although theoretically an excellent remedy to improve the general health, is often contra-indicated, especially early in the disease, on account of its tendency to increase indigestion in many instances. The same remark applies to the use of iron given for the anemia, since

until convalescence is established the iron may increase the digestive trouble and the constipation.

Last of all, the importance of persistence in the treatment must be again emphasized. Unless this is maintained, and especially unless the dietetic treatment is rigorously followed for months, an improvement which has seemed very decided, and even a cure which has seemed complete, will inevitably be followed by relapse.

DILATATION OF THE COLON

This condition may be divided into: (A) Congenital Idiopathic Dilatation (Megacolon congenitum); and (B) Secondary Dilatation.

(A) **Idiopathic Dilatation of the Colon.**—The nature of the disease is not well understood. Writing in 1899 and reporting an instance of the affection¹ I was able to collect but 23 previously published cases which could be accepted with reasonable certainty; although there were a decidedly larger number of others probably incorrectly so named. The number of reported cases has increased greatly since that time. Finney² in a very careful paper collected 206 published articles upon the subject up to that date; Patel³ collected 223 cases, 200 of which had come to autopsy or been operated upon; and since then Porter and Weeks⁴ have found over 100 more. The disease was first described with care by Hirschsprung⁵ and is often called by his name, but a number of well-characterized instances had been published earlier, as, for example, those by Henoch,⁶ Peacock,⁷ Hughes⁸ and still earlier cases go back to Parry⁹ and Billard.¹⁰

Etiology.—The disease is 3 times more frequent in boys. A very probable theory explains the condition, although in a general way, as dependent upon a congenital tendency for the colon to dilate. The cause of this is of unknown nature, and the explanation leaves the matter far from clear. It has been supposed by some to be neuro-muscular; perhaps a paralysis of a region of the colon, with arrest of peristalsis; perhaps a spasm producing functional obstruction. Findings at autopsies do not confirm this view. The disease does not depend upon general muscular atony, since the children are healthy in other respects. The relatively great length of the colon and especially of the sigmoid flexure in infants, to which attention was called particularly by Jacobi¹¹ undoubtedly aids in producing dilatation, but cannot alone account for it, or the disease would be much more common than it is. Whether the hypertrophy of the wall is the cause, the attendant, or the result of the dilatation is uncertain. Unquestionably in some fatal cases in the new born both dilatation and hypertrophy have been found, so that in these instances at least the hypertrophy was not a secondary condition. Generally, however, the latter appears to be secondary to the dilatation.

¹ Amer. Jour. Med. Sci., 1899, Sept.

² Surg., Gynec. and Obstet., 1908, VI, 624.

³ Toulouse méd., 1910, XI, 282.

⁴ Amer. Jour. Dis. Child., 1915, IX, 283.

⁵ Jahrb. f. Kinderheilk., 1888, XXVII, 1.

⁶ Beiträge z. Kinderh., 1861, 123.

⁷ Trans. Path. Soc. of London, 1872, XXIII, 104.

⁸ Trans. Path. Soc. of Phila., 1887, XI, 40.

⁹ Collections from the Unpublished Med. Writings of the late C. H. Parry, 1825, II, 380. Ref. Finney.

¹⁰ Die Krankh. d. Neugeborenen u. Säuglinge, 1829-37, 330. Ref. Finney.

¹¹ Amer. Jour. Obstet., 1869-70, II, 96.

That the dilatation and hypertrophy are both dependent upon a constriction in some lower portion of the gut was maintained by Treves.¹ Careful search at autopsies has, however, failed to reveal any such constriction in the majority of cases; and those where it is found belong more properly to the category of Secondary Dilatation of the Colon.

Pathological Anatomy.—The colon may be involved throughout, or oftener the sigmoid flexure alone. Exceptionally the rectum and the lower portion of the ileum share in the dilatation, which is sometimes enormous (Figs. 254 and 255). Thickening of the intestinal wall, especially of the muscular layer, is present in nearly all instances. The mesocolon may be longer or shorter than normal, and is sometimes thickened. In cases of somewhat long standing inflammatory changes and even ulceration develop.



FIG. 254.—IDIOPATHIC DILATATION OF THE COLON.

Child of 4 years, patient at the Children's Hospital of Philadelphia. No discomfort in the abdomen, enlargement always present, constipation, maximum girth $27\frac{1}{4}$ inches (69.24 cm.). Operation deferred as unwarranted on account of his general excellent health.

Symptoms.—These consist of great dilatation of the colon and of obstinate constipation. In the severer cases the symptoms, or at any rate the constipation, appear in the first few days of life. In others, the congenital *tendency* to dilate is probably present, but, owing to the greater resisting power the evidences of the disease come on more slowly, although still generally in the first 3 months of life, constipation being the first and dilatation developing later and increasing gradually. Occasionally dilatation has not appeared until the age of a year. The constipation is of a most obstinate nature, 1 to 2 weeks or more sometimes passing without a movement. Then under treatment an evacuation of enormous size takes place. The stools are rarely scybalous and in fact may be at times diarrheal, the difficulty of evacuation depending not on any characteristic of the fecal matter, but on lack of power to expel it. The degree of distention varies with the case, but is usually very great. In a child of 2 years and 11 months under my care, the girth equalled 28 inches (71.1 cm.) (Fig. 256). The abdominal distention may be relieved to a considerable extent by a free evacuation of the bowels, but this does not follow in every case. The health is liable gradu-

ally to suffer and emaciation to develop, but this does not occur for several years, and sometimes the condition of general nutrition is but little affected. Vomiting is uncommon, and there is little pain. Dyspnea may be produced by the pressure against the diaphragm. Peristaltic waves may be seen in the colon in cases not too far advanced.

(B) SECONDARY DILATATION OF THE COLON.—The **etiology** of this condition varies with the case. In some instances there has been mechanical obstruction due to a stenosis of the intestine or to obstruction of some

¹ Lancet, 1898, I, 276.

other nature, either in the bowel itself or from without as by the pressure of a tumor or cyst. In other cases habitual constipation may finally be followed by distention of the colon, perhaps as a result of a partial kinking of the bowel through the weight of the fecal matter contained. The disease due to this cause is, however, more liable to occur in later life. An atonic state of the colon with consecutive dilatation may



FIG. 255. —RADIOGRAPH OF IDIOPATHIC DILATATION OF THE COLON.

From the same case as seen in Fig. 254, after the administration of barium. A large, greatly distended loop of colon occupied the right side of the abdomen.

follow some debilitating disease. It is of common occurrence in rachitis, and is a characteristic symptom, to a varying degree, of severe chronic intestinal indigestion and of tuberculous peritonitis.

The **symptoms** of secondary dilatation do not differ materially from those characteristic of the idiopathic variety. The distention in rachitis

is not so great as in instances dependent upon other causes and is seldom productive of such obstinate constipation.

Course and Prognosis of Dilatation of the Colon.—The prognosis of congenital *idiopathic dilatation* is very unfavorable. Of the 24 cases of my report 18 were known to have died and in only 3 was recovery recorded. The fate of the others was unknown. In 59 cases treated medically, collected by Löwenstein,¹ the mortality was 66 per cent.; while in 44 operated cases it was 48 per cent. In a later series of 110 cases subjected to surgical treatment, collected by Terry² the mortality was 25 per cent. The patients rarely live to adult life. The majority die before the age of 5 years of increasing inanition and debility, or some complication as bronchopneumonia, cardiac failure, peritonitis from perforation, or chronic intestinal toxemia. There is not infrequently temporary slight improvement with recurrent exacerbations of the condition. The prognosis of the *secondary dilatation* following stenosis of the intestine is unfavorable, inasmuch as operative interference alone can effect a cure, although life may continue for years if the stenosis is not too great. That of dilatation associated with debilitated health is uncertain and depends upon the severity and the duration of the condition.



FIG. 256.—IDIOPATHIC DILATATION OF THE COLON.

Child of 2 years and 1 month, in the Children's Medical Ward of the Hospital of the University of Pennsylvania. Obstinate constipation from birth, sometimes a week without stool, distention began at 5 months. Maximum girth in hospital $28\frac{1}{2}$ inches (72.37). Right inguinal colotomy performed. Temporary relief was followed by failure of strength and death.

Treatment.—This depends, to some extent, upon the nature of the cause. Cases in which the diagnosis of stenosis can be made demand operation; those the result of debility and digestive disturbance require treatment directed to these conditions. In idiopathic dilatation hygienic remedies should be employed, such as massage of the abdomen, given gently lest ulceration be present; electricity; and measures for the improve-

¹ Centralbl. f. allg. Path., 1907, XXIX, 948.

² Jour. Amer. Med. Assoc., 1911, LVII, 731.

ment of the general health. With this may be combined the administration of strychnine. I have tried pituitrine without benefit. In all forms of dilatation the unloading of the bowels by purgatives and enemata is an unfortunate necessity. This must, however, be done as infrequently as possible, as it tends to weaken still more the muscular power of the colon. High injections are required for this, but often must be accompanied by abdominal massage to assist in expelling the liquid. Puncture of the intestine with a small canula and the drawing off of the gas has been practised in some instances when the distention from this source was great, but is a procedure certainly attended by danger. In all severe cases which have failed to be benefited by other treatment the question of operative interference must be entertained. It should be done seasonably before weakness has become too great; but, on the other hand, it may well be deferred in cases in young subjects in which dilatation and constipation are the only symptoms and the general health is entirely unaffected. The possibility for spontaneous recovery to occur should be permitted, inasmuch as the operation is in itself attended by decided danger to life. As to the choice of operation an artificial anus can be made as a temporary proceeding or resection of the colon performed. The latter is the only satisfactory procedure. In 1 case under my observation, apparently idiopathic, operation consisted in an incision partially through a constriction dependent upon muscular hypertrophy and situated in the upper part of the rectum. Disappearance of the dilatation followed; but this case should manifestly be classified under secondary dilatation.

INTESTINAL OBSTRUCTION

A narrowing or a complete obliteration of the lumen of the intestine may occur in any portion of its course. It may be congenital or acquired, and may be due to many causes. Some of these causes are symptomatic of other conditions and are described elsewhere. In other cases the obstruction appears to be primary, or at least the most important symptom.

1. CONGENITAL STENOSIS OR ATRESIA OF THE SMALL INTESTINE AND COLON

This is a rare condition. Schukowsky¹ observed it but 4 times in 20,000 new-born children. Although possible in any portion of the tube, the stenosis is found oftenest in the small intestine, and much less frequently in the colon. Cowell² collected from medical literature 92 cases of occlusion or stenosis of the duodenum, these including the 57 previously carefully analyzed by Cordes.³ Sometimes only one portion of the intestine is involved; sometimes several. The lesion varies from a mere narrowing to, more commonly, complete atresia, and this may vary from involvement of a very small portion of the tube up to substitution of the entire small intestine by a fibrous cord. In some cases the portions of the intestine above and below the affected region end blindly, the intervening portion having entirely disappeared. The intestine above the obstruction is much distended, that below is collapsed. Malformations elsewhere in the body may occasionally accompany the intestinal deformity.

¹ Ref., *Jahrb. f. Kinderh.*, 1903, LVIII, 323.

² *Quart. Jour. of Med.*, 1912, V, 401.

³ *Arch. of Ped.*, 1901, XVIII, 401.

Etiology.—The cause of congenital stenosis depends upon some pathological process or developmental defect arising during fetal life. The nature of this probably varies with the case. Among those possible are fetal volvulus, peritonitis dependent upon syphilis or tuberculosis, intestinal ulceration, constriction by bands, arrested development, fetal intussusception, and constriction at the umbilical ring. In some instances the stenosis is produced by the pressure by a tumor, or constriction by a Meckel's diverticulum.

Symptoms.—These appear soon after birth, often within a few hours. In atresia or great stenosis they consist of obstinate vomiting, colicky pain, complete constipation except for a few early mucous discharges, and distention of the abdomen, the seat and degree of this last depending to some extent upon the situation of the malformation. It occupies generally the umbilical region, leaving the flanks flattened. When the obstruction is in the duodenum or high in the jejunum gaseous distention is absent, or slight and situated in the epigastrium. The higher the malformation is in the intestine, the sooner does vomiting begin. The vomited matter may eventually be fecal if the obstruction is low in the bowel. The general condition of the child is very serious. The face is pinched, the urine scanty or suppressed, there may be dyspnea from pressure of the gas against the diaphragm, and the temperature is low. If the intestinal stenosis is not complete, the symptoms are of the same nature but less severe.

Course and Prognosis.—The prognosis is always most grave. Death results from collapse, asthenia, or perhaps convulsions. In nearly all cases it occurs in less than a week; very occasionally not before several weeks or even months, if the intestinal stenosis has been less complete. The higher the malformation is in the intestine the shorter is the duration of life. In rare instances, where the stenosis is but slight as shown by autopsy, the patient has lived for some years and even reached adult life.

Diagnosis.—The recognition of congenital obstruction of the alimentary tract is usually not difficult, but to distinguish that of the intestine from that of other regions may sometimes be impossible. In stenosis of the *esophagus* the food is vomited almost at once after swallowing, and the esophageal sound will reveal the obstruction. *Pyloric stenosis* shows the gastric peristaltis; dilatation of the stomach; a pyloric tumor may often be felt; distention is confined to the epigastrium and the vomitus never contains bile. The course of the disease is more prolonged and the symptoms less severe than in cases of intestinal obstruction. Stenosis of the *duodenum* cannot be distinguished from that of the pylorus unless the obstruction is below the entrance of the common bile-duct. In this event bilious vomiting will occur. The vomiting takes place earlier than in stenosis lower in the gut. In stenosis of the lower *ileum* or *colon* the vomiting is later in appearance and is eventually fecal. In all these Röntgenological studies may be of diagnostic value. *Idiopathic dilatation of the colon* cannot be differentiated with certainty from some of the rare instances in which a moderate congenital stenosis of the colon has been present, as in a case reported by Treves.¹ Stenosis of the *rectum* may be detected by local examination. The early and rapid development of the symptoms distinguishes congenital stenosis from that dependent upon fecal impaction, intussusception, volvulus, and the like.

¹ Lancet, 1898, I, 276.

The **treatment** can be only surgical. Operation should be done as promptly as possible, yet the results of this at the early age have been entirely unpromising.

2. CONGENITAL STENOSIS OR ATRESIA OF THE RECTUM OR ANUS

This is very much the most frequent variety of intestinal obstruction, although still uncommon. Several forms of this condition occur, and atresia is more common than stenosis. There may be complete closure of the anus with entirely normal intestine above this. This is the variety most frequently encountered. In a second form not only is the anus closed but the rectum above it exhibits atresia for an extent varying with the case. In still a third variety the anus and the part of the rectum immediately above it are normal but are separated by a membrane from the patulous rectum farther up. In the third form the presence of an accumulation of fecal matter beyond the separating membrane can sometimes be detected by the palpating finger introduced into the anus. In the other varieties this, of course, cannot be done. Any one of the forms may be combined with anomalous communication with the vagina, bladder, or urethra, or exhibit a fistula into the peritoneum or elsewhere. Leichtenstern¹ found such communication in 40 per cent. of the cases. Malformations in other regions of the body may likewise be present.

The **symptoms** are those of intestinal obstruction situated elsewhere, but coming on later than in this latter condition. Inspection or digital exploration of the anal and rectal region will disclose the malformation. Operation should be done as early as possible. This is easy where there is merely an occlusion of the anus or an obstruction of the rectum by thin membrane. Where, however, the rectum is obliterated to any extent it is a more serious matter. An artificial anus must be made in some such cases for temporary relief, leaving for a later period the more difficult plastic operation which will connect the rectal cul-de-sac with the anal region. The plastic closing of any abnormal opening which has been made can be done at the same time. The statistics of the mortality with the various operations which have been employed in 90 collected cases have been carefully analyzed by Ashhurst.²

3. ACQUIRED INTESTINAL OBSTRUCTION

This may be due to various **causes**, some of which must receive separate consideration. A Meckel's diverticulum, (p. 809) or a fibrous cord constituting its remainder or produced in other ways, as by a fetal peritonitis, may sometimes compress the intestine or even strangle it. This may occur at any time in childhood or in adult life. In some cases the intestine is caught in retroperitoneal recesses, or in abnormal openings in the mesentery. Volvulus is another factor uncommon in early life. Peritonitis may cause obstruction, either by the production of fibrous bands, as occasionally seen in tuberculous peritonitis, or by paralysis of peristalsis and the consequent fecal accumulation. Appendicitis acts in a similar manner. Foreign bodies in the intestine, including here especially a fecal impaction, are occasionally causes, as are tumors pressing upon the intestine, and rarely a mass of ascarides in the bowel. Of this last Doberaner³ collected 24 reported instances. Perret and

¹ Ziemssen Handb. spec. Path. u. Therap., 1875, VII, 2, 369.

² Univ. of Penna. Med. Bull., 1907, XX, 96.

³ Prag. med. Wochenschr., 1914, XXXIX, 197.

Simon¹ have added others, including an interesting case observed and figured by them (Fig. 257). An incarcerated strangulated hernia is an occasional cause in children. By far the most frequent cause, however, is intussusception.

The **symptoms** of most of these conditions develop suddenly and are very similar to those in congenital cases, as already described; but the diagnosis of the cause of the obstruction is often impossible. Intussusception and hernia will receive separate consideration.



FIG. 257.—INTESTINAL OBSTRUCTION BY ASCARIDES.

A mass of approximately 40 worms expelled in a case of acute intestinal obstruction in a girl of 8 years. (*Perret and Simon, Jour. Amer. Med. Assoc.*, 1917, LXVIII, 245.)

The **prognosis** is more favorable than in congenital stenosis, since the patient is older and the tolerance for operative interference consequently greater, and since the cause is frequently a removable one.

INTUSSUSCEPTION

Etiology.—The disease constitutes one of the more frequent forms of intestinal obstruction in children. Decidedly over half of the cases occur in the 1st year of life and most of the remainder in the 2d year. It is unusual in the first 3 months, but has been seen as early as the 2d day. In 293 cases collected by Pilz² 158 were in the 1st year. In 314 cases studied by Hess³ 201 were in the 1st year, but only 8 cases in the first 3 months; the 2 youngest in children each 6 days old. Of 397 Danish cases reported by Koch and Oerum⁴ 60 per cent. were in the 1st year and 66 per cent. of these in children of from 4 to 8

¹ *Journ. Amer. Med. Assoc.*, 1917, LXVIII, 244.

² *Jahrb. f. Kinderh.*, 1870, III, 1.

³ *Arch. of Ped.*, 1905, XXII, 655.

⁴ *Mitteilungen aus d. Grenzgeb. der Med. und Chir.*, 1913, XXV, 293.

months. Of their 161 cases occurring after the age of 1 year, all but 30 were not over 6 years of age. The disease is from 2 to 3 times more common in males than in females. The occurrence of diarrheal conditions occasionally predisposes, as does constipation and colic; but in most instances intussusception develops in those who have been in apparently perfect health. Abnormal conditions of Meckel's diverticulum have been reported as the cause in a number of instances. I have reviewed this subject elsewhere.¹ The vermiform appendix may in rare instances invaginate itself and be the cause of intussusception of the intestine (Monsarrat).² Corner³ was able to collect 16 reported cases of this occurrence. Injuries of the abdomen are also to be noted as occasional causes. The presence of large fecal masses or of polypi tends to produce invagination through the efforts of the intestines to expel them. Finally *agonal intussusception* is of quite frequent occurrence in infancy, caused by irregular peristalsis of the intestine developing in the few moments preceding death.

Pathology and Pathological Anatomy.—The condition consists in an invagination of one portion of the intestine into another, the result of irregular contraction and peristalsis of the intestinal walls. The invagination is descending in type, an ascending invagination being very rare. The upper portion, the *intussusceptum*, slips into the lower, the

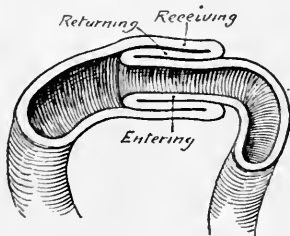


FIG. 258.—DIAGRAMMATIC REPRESENTATION OF THE PRODUCTION OF INTUSSUSCEPTION. (Kemp, *Diseases of the Stomach, Intestines and Pancreas.*)



FIG. 259.—INTUSSUSCEPTION.

Intussusciptions to the left. Courtesy of Dr. Robert LeConte.

intussusciptions, pulling the mesentery with it. The intussusception thus consists of three thicknesses of intestinal wall, two mucous surfaces being in apposition, and two serous likewise (Fig. 258). The dragging upon the mesentery makes the intestine assume a curved, sausage-shaped form, with the concavity toward the mesenteric attachment to the spinal column (Fig. 259). Although the bowel can remain patulous and the circulation

¹ Jour. Amer. Med. Assoc., 1914, LXII, 1624.

² Liverpool Med.-Chir. Jour., 1901, XXI, 68.

³ Annals of Surg., 1903, XXXVIII, 690.

of blood in it be preserved, this is usually not the case, but swelling promptly begins as a result of compression of the blood-vessels. This not only produces complete intestinal obstruction but an incarceration of the intussusception, which later becomes irreducible through the adhesive inflammation between the adjacent serous surfaces. The mucous membrane is deep-red, especially the apex of the intussusceptum, and finally strangulation takes place with more or less complete death of the part. In the fortunate, but rare, cases this may become entirely separated by the gangrenous process, and discharged through the anus, while the adhesions formed between the bowel and the neighboring tissues and between the contiguous coats of the bowel itself serve to reestablish the lumen of the gut.

The intussusception does not, however, become irreducible through adhesive inflammation for several days and sometimes even a week. Previous to this it is the swelling which prevents the intestine resuming its normal position. The time when gangrene begins varies greatly, it developing more frequently and sooner in the acute cases. The intestine above the intussusception is generally dilated, that below it contracted.

Intussusceptions are classified according to their situation:

1. **Ileac (enteric) intussusception** affects the small intestine in any part, and usually this only. It is less often seen than some other forms in early life, but one of the more common after the period of childhood. This does not apply to the *agonal* variety which is not uncommon in infancy, involving as many as from 6 to 12 different positions in the small intestine, being most frequent in the jejunum. Agonal intussusception shows no evidence of inflammation or swelling, and is without clinical significance.

2. **Colic intussusception** may affect any part of the colon. It is one of the more common varieties, yet not as often seen as the ileocecal.

3. **Ileocolic intussusception** exhibits invagination of the ileum through the ileocecal valve. The cecum may then be invaginated secondarily, but the ileum remains as the most prominent protruding portion. This is the least frequent variety of intussusception.

4. **Ileocecal intussusception** is the form oftenest seen, and the younger the child the more likely is the invagination to be of this sort. In this the cecum with the ileum behind it passes into the colon, the valve continuing to be the apex of the projecting portion.

Besides these types mixed forms of various sorts may occasionally occur, including instances of double intussusception. The relative frequency of the four main types, as occurring in children, seems to vary somewhat according to the statistics given, yet the differences are not great. This may be seen, for instance, in the 180 cases of Leichtenstern¹ under 10 years of age and the 380 cases of Koch and Oerum.²

TABLE 81.—FREQUENCY OF INTUSSUSCEPTION, LEICHTENSTERN, 180 CASES

Variety	Under 2 years, per cent.	2-5 years, per cent.	6-10 years, per cent.
Ileocecal	70	49	41
Ileac	6	13	38
Colic	19	25	21
Ileocolic	4	13	0

¹ Prag. Vierteljahrsch. f. prakt. Heilk., 1873, CXVIII-CXIX, 188, et seq.

² *Loc. cit.*; 301.

TABLE 82.—FREQUENCY OF INTUSSUSCEPTION. KOCH AND OERUM. 380 CASES

Variety	Under 1 year, per cent.	Over 1 year, per cent.
Ileocecal.....	49.5	38.0
Ileac.....	2.6	9.2
Colic.....	12.0	23.7
Ileocolic.....	3.5	1.3
Mixed.....	3.9	1.9
Undetermined.....	28.0	25.7

Koch and Oerum maintain that the larger proportion of the cases called "undetermined" could with propriety be placed among the ileocecal.

As time passes the intussusception of all varieties is prone to increase in size, the apex always remaining unchanged and the intussusciens invaginating more and more. As a result the ileocecal form may finally occupy all of the rectum, and the apex of the intussusception nearly reach the anus or even pass through it.

Double intussusceptions are less frequent, probably over 90 per cent. being single (Corner).¹ For instance, an enteric intussusception may extend downward to the cecum, and then push the cecum before it, producing a secondary ileocecal invagination. Double intussusception of other forms may occur.

Symptoms.—With or without previous evidence of intestinal disturbance the child is suddenly attacked by severe pain, vomiting, restlessness and great prostration. The face is pinched, the eyes sunken, the pulse feeble and rapid, the urine scanty, the temperature normal or below it. The abdomen is at first soft; later distended and often tender. The fecal matter present in the colon and rectum is evacuated during the 1st day, but after this fecal movements are very small in amount or more often entirely absent, and little if any gas is passed by the bowel. Pain comes on in paroxysms, attended by straining efforts if the intussusception has reached the rectum, and by the frequent passage of small quantities of blood-stained mucous, or even of blood in considerable quantity. Blood generally appears in the first 12 hours, but sometimes not for 1 or 2 days. This condition of the bowel movements is one of the most characteristic, but may be wanting in some cases. Absolute failure of evacuation of fecal matter and of gas is most common in enteric invagination, and in these cases, too, hemorrhage from the bowel may be later in appearing, or absent entirely. Although vomiting continues very persistent and frequent it is always a less-marked symptom than in other forms of intestinal obstruction. It is usually worst at the beginning, but may continue severe throughout the attack; or it may sometimes occur only a few times daily. It is most marked in enteric intussusception. Stercoraceous vomiting is seen in only a small proportion of cases and generally not until late. Pain, too, is a variable symptom. Sometimes intensely severe and causing loud outcries, it may be evidenced only by the grunting sounds which attend the accompanying tenesmus. It is usually most intense early in the attack. In children old enough to describe it, it may be localized in the region of the intussusception or about the umbilicus. A tumor is discoverable in the majority of instances. It can often be found very early in the disease, even on the 1st day, and may reach the anus by the 2d day, although

¹ *Loc. cit.*, 690.

usually later than this. It can very frequently be felt in the rectum and then gives to the finger a sensation very like that of the vaginal portion of the uterus, the lumen of the bowel corresponding to the os. Koch and Oerum¹ maintain that a tumor was found by abdominal palpation or by rectal examination in 85.5 per cent. of their 380 cases. As to the position occupied, as shown by abdominal palpation, it was upon the left side, either the upper or lower quadrant, in over 50 per cent. of the cases. In about 33 per cent. the tumor had been discovered by rectal examination. During the paroxysm of pain rectal palpation shows the tumor temporarily increasing in size and approaching nearer to the anus. When protruding from the anus it may have the appearance of a rectal prolapse or a polypus or hemorrhoid. Felt through the abdominal walls between the paroxysms of pain the tumor may occupy various positions depending upon the seat of the intussusception. In the commoner ileocecal form it is situated usually in the region of the descending colon curving around the umbilicus from above to the left side. In the ileocolic form the tumor is upon the left side extending from the costal border downward, but without arching about the navel. In enteric intussusception a tumor can seldom be detected.

The symptoms described are those characteristic of most acute cases. At times, however, instances occur in which many of the symptoms are so little marked that the disease may be entirely overlooked if sufficient attention is not paid to the condition of the bowel-movements and the discovery of a tumor. Occasionally cases are more subacute in nature, the onset being more gradual, the pain and vomiting less severe and constipation less complete. Still other exceptional cases, seen only in older children, are of a more chronic nature, in which the occlusion is not complete and the circulation in the mesentery not entirely shut off. There is little pain or vomiting, no definite intestinal symptoms, and no bloody, mucous movements. Diarrhea may replace constipation. The symptoms may come in attacks separated by intervals of comparative health, and suggest recurrences of catarrh of the large intestine. The patients gradually lose strength or may suffer suddenly from symptoms of strangulation, as in an acute attack. Only the discovery of a tumor makes a diagnosis possible.

Course and Prognosis.—If the reduction is accomplished the tumor can no longer be felt, and all the other symptoms rapidly disappear. If this has not taken place the course of the disease is progressively onward to a fatal issue. A few exceptions are seen in which spontaneous reduction occurs, or in which gangrene and subsequent discharge of the invaginated bowel take place and the patient survives the septic condition and risk of perforation. Cicatricial stenosis may develop later in such instances.

Death is the result generally of collapse from the shock which the intestinal lesion produces. In the more subacute or chronic cases it follows from increasing exhaustion, sometimes with a final rise of temperature to a considerable height. Death from peritonitis is not common. The average duration of acute fatal cases without operation is a week or sometimes less, with a range of 1 day up to 2 or 3 weeks. Chronic cases may last for months.

There is often seen a disposition for the intussusception to *relapse* after successful reduction. This takes place with greatest frequency on the first day after reduction has been accomplished, but may be

¹ *Loc. cit.*, 338.

much delayed. It sometimes happens that this occurrence takes place several times. Some of these cases are certainly mistaken instances of relapse; it being supposed that reduction has occurred, when, in reality, this did not happen. Undoubtedly relapse is possible, although not very frequent. Koch and Oerum¹ found 10 such cases in their list, with 9 other doubtful ones and 6 instances of recurrence at later periods.

The *prognosis* is always grave especially in acute cases, although less so than formerly. The younger the patient the more serious the condition. Untreated cases are practically always fatal. The probability of recovery varies, too, directly in proportion to the quickness with which the diagnosis is made and treatment instituted. In general the average mortality at the present day, including cases not operated upon, may be placed at 60 to 70 per cent. In the 314 cases reported by Hess² 211 recovered and 103 died. Leichtenstern's³ figures gave a total mortality of 73 per cent. for 557 cases of all ages. In the 1st year the mortality was 86 per cent. The death-rate has lessened since operative interference has become more common and the technique better. Thus Weiss⁴ in 322 collected cases of all ages found the mortality in infancy (177 cases) without operation, 84 per cent.; with primary laparotomy, 39 per cent.; with secondary laparotomy after unsuccessful treatment of other sorts, 46 per cent. In childhood (85 cases) without operation, 78 per cent.; in primary laparotomy, 10 per cent.; in secondary laparotomy after other unsuccessful treatment, 50 per cent. The chance of recovery depends, too, upon the promptness with which operation is performed. Gibson⁵ in 187 operative cases, collected since antiseptic methods have improved, shows this well, the mortality in patients operated upon on the 1st day being 37 per cent.; on the 2d day, 39 per cent.; 3d day, 61 per cent.; 4th day, 67 per cent.; 5th day, 73 per cent.; and 6th day, 75 per cent.

Diagnosis.—This usually presents little difficulty. The cardinal diagnostic symptoms are the sudden development of abdominal pain, vomiting, tenesmus and bloody stools without fecal matter, prostration and absence of fever. The diagnosis is made certain by the discovery of a tumor. Perplexities, however, arise at times and mistakes are far from uncommon. The bloody, mucous movements may be mistaken for *colitis*. Conversely colitis may be supposed to be intussusception, and I have seen operation urged accordingly. The earlier occurrence of diarrhea and the presence of fecal matter and especially fever, aid in the recognition of the inflammatory disorder. *Henoch's purpura*, with hemorrhage into the lumen and the walls of the intestine, may strongly suggest intussusception, but the presence of evidences of purpura elsewhere aid in distinguishing it. It is possible, however, for purpura to be attended by intussusception, and a number of such cases have been reported. The protruding of an intussusception from the rectum accompanied by straining and the passing of mucus may sometimes strongly suggest *prolapse of the rectum*. I have known the two conditions to be combined. As stated, the discovery of a tumor usually removes doubt, but as this is often difficult, especially through the abdominal wall, in all questionable cases a careful examination should be made under anesthesia.

¹ *Loc. cit.*, 326.

² *Loc. cit.*

³ *Loc. cit.*

⁴ *Centraltbl. f. d. Grenzgeb. d. Med. und Chirurg.*, 1899, II, 702.

⁵ *Arch. of Ped.*, 1900, XVII, 99.

Enteric intussusception seldom has a discoverable tumor and there is no tenesmus, and these cases cannot with certainty be distinguished from instances of acute intestinal obstruction from other causes. It is to be remembered also that *appendicitis* is attended by the development of a tumor and often by constipation. There are, however, no bloody stools and tenesmus, and the tumor occupies usually the region of the cecum, is more superficial, and of a different shape. In typical cases of intussusception, the injection of bismuth and the taking of an x-ray picture may be of diagnostic aid, as in a case reported by Snow and Clinton.¹

Treatment.—As soon as the diagnosis is made in cases which have lasted only 1 or 2 days determined efforts may be made to reduce the intussusception by other than surgical measures. The child should be anesthetized, the hips elevated, and the patient now and then held inverted. Injections should meanwhile be given of either air, water, or oil. There is probably no great danger of rupture of the intestine with proper precautions, but the pressure with air can be less accurately controlled than that with liquid. Air may be injected from a bulb-syringe or hand-bellows attached to a catheter. The anus must be compressed around the tube by the fingers, and the injection given slowly and carefully. In place of this, oil, or a warm normal saline solution, may be employed. The liquid should be in a fountain syringe which may be elevated not over 6 feet (173 cm.) above the bed on which the child lies; the pressure being maintained for 15 or 20 minutes. If successful, a rumbling sound can sometimes be heard; the tumor entirely disappears, very careful examination being necessary to make sure of this; the distention produced by the gas or liquid employed ceases to exist; the aspect of the child improves; vomiting stops; and a fecal stool may shortly occur. If unsuccessful, or if the result is uncertain, the liquid or air must be allowed to escape and the procedure tried once again. If still there is no success, operative aid must be had *immediately*, and the intussusception found and reduction accomplished by withdrawing the invaginated portion if this is possible. In fact, owing to the frequent impossibility of determining accurately whether reduction has been accomplished, or of knowing what may be the condition of the intestinal wall, it is safer to regard all cases as purely surgical, and to proceed at once with operation for reduction, without attempting any medical treatment. In cases coming late to operation adhesion and swelling may prevent reduction; or the condition of the bowel may be such that enterostomy is necessary, and either an artificial anus must be made, or a resection of the intestine performed.

On account of the danger of recurrence of the trouble after either mechanical or operative reduction, peristalsis should be quieted as far as possible by small, repeated doses of an opiate. A purgative should never be administered.

HERNIA

Hernia in children is of several varieties. Those deserving special mention are: (1) Umbilical; (2) Inguinal; (3) Diaphragmatic, and (4) Ventral. Of these the umbilical and inguinal are far the most frequent. Femoral hernia is so uncommon that further reference need not be made to it. The rare internal hernias other than the diaphragmatic will also be omitted.

¹ Amer. Jour. Dis. Child., 1913, VI, 93.

1. UMBILICAL HERNIA.

Hernia of the intestine at the umbilicus may be either (a) congenital or (b) acquired.

(a) **Congenital Umbilical Hernia** (*Hernia into the Cord*).—This is a very uncommon condition; of which I can recall seeing not more than 2 instances. Lindfors¹ in 20,735 births found it present in the ratio of 1:5184. The hernia forms a tumor, oval, round or conical in shape, and of the size of a walnut up to that of an orange or larger. The sac appears to be composed of the distended umbilical cord, its walls consisting only of peritoneum and of the amnion of the cord, and being of a greenish-white color and transparent character. The contents are usually coils of intestine, but sometimes the stomach, the spleen, or all or part of the liver, may be found in it. The color of these is readily distinguishable through the sac-walls. The size and tension of the mass increase with crying or coughing. The hernia can sometimes be reduced, sometimes not; and when of small size recovery may take place spontaneously with the process of the separation of the cord. In this event a reactive inflammation sets in around the ring-shaped border of the hernia, the color of the sac changes, and the umbilical cord shrinks and finally it and the amnion separate. Granulations then spread gradually over the surface remaining, generally with free suppuration. As the wound thus left heals and shrinks the hernia disappears within the abdominal cavity. A cicatrix remains but no real navel.

Many dangers, however, attend this process and a fatal issue generally results. Peritonitis is very liable to occur, the hernia becomes gangrenous or general sepsis develops. The **prognosis** is on the whole grave. By far the larger proportion of patients died until the radical operation was introduced and perfected, and even still the mortality is high. In the case of very large hernia, with the presence in the sac of a considerable portion of the abdominal contents, the continuance of the child's life is scarcely possible. In 91 instances of hernia of the cord, collected by Lotheisen² the mortality in the 68 operated cases equalled 29.4 per cent., and in the 23 unoperated cases 65.22 per cent. Somewhat similar statistics are given by Safford³ with a mortality of 33 per cent. in 73 operated cases, and 53 per cent. in 15 unoperated cases. Sometimes the sac breaks during birth, leaving the child partly eviscerated. Very often other malformations are present. Not infrequently the infants are premature or still-born.

The **diagnosis** is readily made except in the case of small cylindrical hernias into the cord. Here it is easy to overlook the condition and to apply a ligature, which, of course, ligates the intestine as well. Every child born with the cord decidedly swollen close to the body should be examined very carefully before a ligature is applied.

Treatment consists, first of all, in the greatest care in handling a congenital hernia and in the use of every possible antiseptic precaution. If the rupture is small and if reduction can be made quite easily, this may be done and an antiseptic compress applied and attached with adhesive plaster. The child must not be lifted into an upright position until the wound has completely cicatrized, and this may require weeks. If the hernia is not reducible, it may be covered with an antiseptic protective dressing in the hope that granulations may form and the process go on

¹ Volkmann's Sammlung klin. Vorträge, 1893, n. s. 63, Gynac. No. 26, 624

² Wiener klin. Rundschau, 1903, XVII, 757.

³ Phila. Med. Jour., 1901, VII, 393.

as just described. A much more successful plan of treatment, however, applicable also to the small, reducible hernias, is the performing of a radical operation as soon after birth as possible and without any previous efforts at reduction being made. The operation may be done either with or without the opening of the peritoneum. The temperature of the child should later be maintained by the use of external heat as in the treatment of premature infants.

(b) **Acquired Umbilical Hernia.**—This is a very common and seldom serious affection of infancy. It generally develops in the first few months of life, and is seen most frequently in thin children or in those with indigestion and flatulent distention of the abdomen from other



FIG. 260.—A MILD DEGREE OF ACQUIRED UMBILICAL HERNIA.

(Hecker and Trumpp, *Atlas of Diseases of Children*, American Translation, Fig. 22, p. 82.)

causes. Infants in whom there is much crying or such pressure as may attend constipation are also predisposed to it. The influence of phimosis through straining efforts is considered very questionable. The hernia appears oftenest as a small elastic tumor covered with skin, not sensitive to pressure, varying in size from a simple convexity of the navel to a tumor the size of a small marble, or occasionally larger, and globular or irregular in shape according to the size and form of the opening. Frequently it cannot be detected at times, and it is always reducible unless the child is crying or straining. Strangulation very rarely occurs. The hernia consists of small intestine which protrudes through a portion of the umbilical ring, and is covered by the abdominal parietes. The prognosis is almost entirely favorable. The majority of cases will recover spontaneously if such causes as continuous abdominal distention or persistent straining efforts be removed. Yet as it is possible for the hernia to persist, and in view of the fact that the longer the opening remains the more likely is this to happen, every case should receive

treatment. The **diagnosis** presents no difficulty. A large serous accumulation in the abdominal cavity could distend and project through the centre of the navel, but the attending symptoms would remove all question. Preventive **treatment** is important. A firm compress should be worn under the abdominal band for the first few months, and all conditions liable to produce hernia should be removed. If a hernia is present it should be kept constantly reduced until the opening in the abdominal wall has had time to close. Usually it is quite sufficient to draw the skin into two folds, one on each side of the hernia and meeting over it; holding these in place by straps of adhesive plaster crossing over the navel, or by a broad horizontal band of adhesive plaster reaching to the lumbar regions (Figs. 260, 261). Another method is the following: A silver quarter of a dollar is laid upon the adhesive surface of a piece of rubber adhesive plaster 2 inches square; over this is placed the broad strap referred to, with its adhesive surface next to that of the smaller



FIG. 261.—BAND OF ADHESIVE PLASTER OVER AN ACQUIRED UMBILICAL HERNIA.—THE PLASTER IS TENSELY DRAWN AND APPLIED AND FASTENED OVER THE RIBS ON BOTH SIDES, SO THAT A LONGITUDINAL FOLD OF THE ABDOMINAL WALL IS DRAWN OVER THE HERNIA.

(Hecker and Trumpp, *Atlas of Diseases of Children*, American Translation, Fig. 23, p. 83.)

piece. After reducing the hernia and pressing the sides of the abdominal walls slightly together the band is applied with the quarter dollar directly over the position of the navel. My own preference is for a simple adhesive band without the use of the coin. The dressing should be worn constantly, changing it from time to time as the old one loosens. The dressing must, of course, not be removed during the bath. Several months are required before the opening is permanently closed. Occasionally the plaster produces a great deal of cutaneous irritation, especially in the first few months of life. The employment of zinc oxide plaster tends to avoid this difficulty. When the irritation is obstinate, treatment may be deferred for a while until the child is a little older; or, if the hernia is very small, it may be possible to trust the cure to Nature. When a dressing is required and plaster cannot be worn, we must depend upon a closely applied bandage of woollen material or webbing, although this is less satisfactory. In no case should any apparatus be used with a rounded surface which pushes the hernia inward. The pressure from without merely serves to keep the umbilical ring open.

Hernia in children older than a year resists mechanical treatment obstinately. Such an appliance as described may be tried; and if it seems to do good its employment must be persisted in for months. If cure is not then progressing, a radical operation is indicated.

2. INGUINAL HERNIA

This is much less frequent than umbilical hernia in the first few weeks of life, but more common when developing after this period. As in the umbilical affection, it may be congenital or acquired. The great majority of the acquired cases depend, however, on favoring conditions which are congenital in nature; viz. a patulous state of the funicular process of the peritoneum through which the testicle descends; the shortness and straightness of the canal, and the width of the inner ring. Omitting, then, the truly acquired hernia of later childhood, which is identical in nature with that of adult life, and less often seen, we may divide the hernias of infancy into (a) **congenital hernia of the tunica vaginalis**, in which the funicular process of the peritoneum is completely open and the intestine descends to and often surrounds the testicle; (b) **funicular hernia**, in which the tunica vaginalis is closed above the testicle and the intestine fills the funicular process down to this closure, the intestine in this variety not enveloping the testicle; (c) the **encysted or infantile hernia**, a rare form in which the internal ring has closed but the intestine pushes down a pouch of peritoneum beside this or into the patulous funicular process below the ring. Except for the differences mentioned, which are purely pathological, these various forms cannot with certainty be distinguished from each other except at operation, and then the matter is of no practical moment.

Etiology.—Apart from the anatomical causes mentioned, age is a strongly predisposing factor, the majority of cases in infancy occurring in the 1st year of life, but sometimes not until later, and sometimes seen immediately after birth. Heredity, too, plays some part. The great majority of instances are met with in boys, 85 out of 94 cases reported by Ashby and Wright¹ being in this sex. Distention of the abdomen by gas, excessive crying or coughing, straining at stool the result of diarrheal disturbances, the straining on urination caused by excessive phimosis or urinary concretions, impairment of the general health, and similar conditions may constitute the final active cause.

Symptoms.—These differ little from those of adult life. The rupture is oftener situated on the right side, but not infrequently is double. The contents of the sac are much the same as in the adult. They consist usually of small intestine only, perhaps with omentum; while sometimes the cecum and vermiform appendix occupy the sac, rarely Meckel's diverticulum, and occasionally an ovary. In a considerable number of cases the appendix is found within the hernial sac, either alone or with other portions of the intestinal tract. The literature of this condition has been reviewed by Jopson.² Reduction is generally easier than in adult cases, as the hernia is usually smaller and adhesions have not formed. It is only occasionally that a large rupture fills the scrotum (Fig. 262).

Prognosis.—The prognosis of the disease is favorable, complete recovery usually following the early application of a suitable truss; or

¹ The Dis. of Child., Amer. Edit., 1893, 136.

² Univ. Med. Magaz., 1900, XIII, 94.

if this does not succeed, the radical operation giving excellent results. Strangulation is comparatively uncommon in early life. It is seen oftener in the first 2 years than in childhood after that period. Estor¹ in a study of 207 cases of strangulation in infants up to the age of 2 years, estimates that the likelihood of the development of this as compared with that of adults is only in the ratio of 1:131. Strangulation according to Moynihan² is more prone to occur in the first 3 months than after that period during the 1st year. Whitelocke³ reported 2 cases in infants of 17 and 22 days respectively.

Diagnosis.—The only difficulty in diagnosis is in distinguishing the lesion from *hydrocele*, the two conditions often closely resembling



FIG. 262.—LARGE INGUINAL HERNIA.

Infant of 14 months, in the Children's Hospital of Philadelphia.

each other. Hernia is usually opaque with transmitted light and hydrocele translucent; but this is open to exceptions and hernias may also sometimes appear translucent, if bowel only is present and is distended by gas without fecal matter. Hydrocele is dull on percussion and reduces slowly and often not at all. Hernia gives an impulse on coughing and reduces more quickly and often with the characteristic gurgling sound. The difficulty in diagnosis is increased by the fact that a hydrocele may occupy the tunica vaginalis and a hernia of the cord be situated immediately above this. Strangulation may in exceptional cases be readily confounded with severe colic unless the possibility of this occurrence is borne in mind and a systematic examination made. I have seen this

¹ Rev. de chir., 1902, XXV, 249.

² Lancet, 1897, II, 788.

³ Brit. Jour. Child. Dis., 1913, X, 253.

error made in a child of less than a year. In later childhood the ordinary symptoms of hernia develop, and the diagnosis presents no difficulty.

Treatment.—This is very satisfactory in that the majority of cases will recover completely under the application of a suitable truss and the removal of the exciting causes. A truss of hard rubber or a skein of woolen yarn must be worn constantly, and the mother impressed with the importance of never allowing the hernia to descend. The skin under the truss must be kept dry and clean and in a healthy condition. The constant wearing of a truss will often cure a rupture in 3 months in cases occurring in the 1st year of life; a longer time is required if treatment is begun after this period.

When the use of a truss does not succeed by the end of the 1st year, and in every case where the hernia is found irreducible after gentle efforts, a radical operation for permanent cure must be employed. This should be done promptly in irreducible cases even though no threatening symptoms are present. It has also been recommended to close the opening of the sac by the injection of paraffin about it.

3. DIAPHRAGMATIC HERNIA

Omitting hernias of this nature which result from severe trauma, as from wounds of the diaphragm, this unusual condition is generally congenital; and even in the occasional acquired cases seems then dependent upon an already existing congenital defect in the diaphragm. The intestine, and often other viscera as well, project to a greater or less degree into the thoracic cavity through an abnormal opening in the diaphragm. In well-marked cases physical examination shows the stomach and a large portion of the intestine in the pleural cavity, with displacement of the lungs and heart and an abnormal sinking in of the abdominal walls. The percussion note in the thorax is tympanitic; the respiratory murmur absent. There are also dyspnea, cyanosis, and vomiting and other digestive disturbances. Often, however, no diagnosis is made until at a post-mortem examination. Occasionally subjects of this condition live until adult years, but more frequently the severity of the symptoms terminates life in the 1st year, or death follows incarceration or strangulation with the usual manifestations. The only treatment possible is to guard against incarceration by careful diet and hygiene, and to operate immediately should this accident occur.

4. VENTRAL HERNIA

This is a not very common form of hernia. It consists in the protrusion of a small portion of intestine either through a defect in the median line of the abdominal wall or in the lumbar region (*lumbar hernia*). The former is always small, sometimes multiple and is usually accompanied by umbilical hernia. The rare lumbar hernia may reach a much greater size. Treatment in either case is very similar to that recommended for umbilical hernia.

INTESTINAL ULCERATION

This is the result of so many causes, and is a symptom of such diverse conditions that no more than a mere reference can be made to most of the forms in this connection, only a few being treated of here more in detail.

(1) DUODENAL ULCER

The round peptic ulcer, similar in nature and in method of production to that occurring in the stomach, is of uncommon occurrence in children, yet distinctly more frequent than was formerly supposed. Schmidt¹ found it present in 20 out of 1109 autopsies in the 1st year of life; *i.e.* 1.8 per cent.; and in 17 out of 2715 autopsies in children from the 2nd year onward; *i.e.* 0.6 per cent. In a previous publication² I reported 2 cases and collected a number of instances occurring in medical literature, and Holt³ and Veeder⁴ have each reviewed the subject carefully. The disease is oftenest seen in atrophic infants in the 1st year of life. It occurs perhaps most frequently in the new born and is then a cause of melena. The ulcer is usually single, although two or more are occasionally seen; possesses the sharply defined edges characteristic of gastric ulcer; and is generally situated on the posterior wall of the duodenum and above the papilla. It may involve only the mucous membrane, or may extend to the serous layer and may even perforate. Gerdine and Helmholz,⁵ in reporting 11 personal cases, support the view that the condition may be epidemic and is dependent upon the action of the streptococcus viridans.

Symptoms.—In a large proportion of cases these are entirely lacking, and the condition is purely a post-mortem finding. In others there may be a sudden fatal collapse, indicating a possible concealed hemorrhage or an intestinal perforation, but without sufficient data to render such a diagnosis possible. Melena in the new born may, as stated, be dependent upon duodenal ulceration, but there is usually no possibility of determining this with certainty, and the majority of cases of melena are not produced in this way. The only truly suggestive symptoms indicating duodenal ulceration are hematemesis and the passage of blood by stool. The blood may be in large amount and bright-red, or it may appear as coffee-ground vomiting and as tarry evacuations. In older children there may sometimes be pain and tenderness in the region of the duodenum just below the liver to the right of the median line; but this is an unusual symptom. In a number of instances there have been seen symptoms suggesting pyloric stenosis, depending probably upon pylorospasm produced by a reflex irritation from an ulcer just below the pylorus. Such cases have been reported by Finney,⁶ Torday⁷ and others.

The **prognosis** is very uncertain. Death may result promptly from hemorrhage; or a temporary improvement of symptoms may be followed by relapse. Recovery seems possible, but is a matter not susceptible of proof, and is probably not of frequent occurrence.

The **diagnosis** can be made only provisionally. The preponderance of the passage of blood by the bowel over hematemesis is suggestive.

Treatment.—This consists in efforts to check hemorrhage or to prevent recurrence. For the first, trial may be made of gelatine and of epinephrine internally. The patient must be at absolute rest, and the diet of the lightest and most unirritating sort, at first given by enema only. An ice-bag may be placed over the region of the duodenum, guarding carefully against depression if the patient is an infant. If hemorrhage is severe and is unchecked by other measures, or if symptoms of a perforative

¹ Berl. klin. Woch., 1913, L, 593.

² New York Med. Jour., 1911, Sept. 16.

³ Amer. Jour. Dis. Child., 1913, VI, 381.

⁴ Amer. Jour. Med. Sci., 1914, CXLVIII, 709.

⁵ Amer. Jour. Dis. Child., 1915, X, 397.

⁶ Proc. Royal Soc. of Med., 1908-9, Sect. for Dis. of Child., 67.

⁷ Jahrb. f. Kinderh., 1906, LXIII, 563.

peritonitis develop, exploratory laparotomy is the only course remaining open. To prevent the return of hemorrhage the diet must continue light and free from substances of an irritating nature, such as spices or food containing much waste material. Over-exercise must be avoided, especially such as would produce undue strain or pressure upon the abdominal region.

The **ulceration of ileocolitis** is a very common condition in infancy. The ulcers are very abundant and of sizes varying from minute erosions to larger, deeper lesions. Further description will be found under the heading of this disease.

Typhoid ulcers are, as a rule, not nearly so common or so large in early life as later. They may sometimes, however, be abundant and extensive even at this period, and perforation may take place. (See Typhoid Fever, p. 390.)

Syphilitic ulceration of the intestine is a rare occurrence in children and infants. It is the result of gummatous or necrotic alteration affecting the intestinal canal.

(2) TUBERCULOUS ULCERATION

(Tuberculosis of the Intestine)

The method of infection of the intestines by tuberculosis and the frequency of this have already been described under the heading of Tuberculosis, where some statistics bearing upon the subject will be found (pp. 543, 546 and 557). The disease may be primary in the intestine, but is usually secondary to lesions in the lungs, and is nearly always combined with involvement of the mesenteric lymph-glands. It may occur at any period of early life, but is most frequent in early childhood, yet less common than other forms of tuberculosis at this time.

Pathological Anatomy.—The lesions are situated chiefly in the small intestine, especially the ileum near the ileocecal valve; although they occur to a less extent in the cecum, colon, jejunum, and the appendix. The serous membrane of the bowel may be covered by tubercles in acute miliary tuberculosis, but as a rule the lesions as discovered at autopsy are of an ulcerative nature, situated in the mucous membrane and submucous layer. Early in their course they consist of miliary nodules, soon becoming of a yellowish color, and often numerous and widespread. Some of these break down and form small erosions, and then may extend and coalesce, forming large ulcers of even 1 to 2 inches (2.5 to 5 cm.) in diameter. The number of these larger ulcers is usually not great. In its typical form the tuberculous ulcer is of irregular shape with uneven infiltrated edges which project above the level of the surrounding mucous membrane, while miliary tubercles cover the bottom. The largest diameter is usually transverse to the canal of the intestine, and sometimes, in elliptical form, the ulcer may reach nearly or quite around the lumen. The smaller ulcers show a loss of mucous membrane only; the larger ones penetrate the submucous tissue as well, and even to, or sometimes through, the serous layer. If the case is long-continued some ulcers exhibit at autopsy cicatricial changes, with healing of the lesion and consequent contraction of the intestine at this position. Perforation into the peritoneal cavity is uncommon, because of involvement by the tuberculous process of the peritoneum adjacent to the ulcer, and consequent formation of adhesions.

Symptoms.—These are far from characteristic, especially in the early stages. Small tuberculous ulcers of the intestine frequently produce no symptoms whatever and are discovered only at autopsy. In other instances the symptoms are those of ileocolitis. In such cases the possibility of the lesions being tuberculous may be suspected from the chronicity of the case, and from the association of undoubted evidences of the infection elsewhere in the body, especially in the lungs. The stools are liable to be more watery in character than in ileocolitis, offensive, and to contain more or less blood, especially in older children. Abdominal distention and tenderness may be present; there is irregular fever; wasting is often great; anemia and debility decided; appetite is diminished; the pulse weak, and the abdomen tympanitic. In advanced cases the symptoms of tuberculous peritonitis are often present also, or deep palpation may reveal enlargement of the mesenteric lymphatic glands. Microscopic examination may sometimes show tubercle bacilli in the stools; yet the possibility of these coming from the swallowing of tuberculous sputum must not be forgotten.

Course and Prognosis.—The disease may run an irregular course and last for months, with a constantly increasing loss of health, or with temporary periods of improvement, diarrhea perhaps alternating with constipation. Although recovery is probably possible it is certainly very uncommon, and the majority of patients with the disease gradually fail in health and die from exhaustion, often with a terminal marantic edema. In other cases death may occur from some complication, such as profuse hemorrhage, peritonitis, or tuberculosis of some other region.

Diagnosis.—This depends chiefly upon the association of chronic intestinal derangement with tuberculosis elsewhere in the body, the very slow development of symptoms at the beginning, and the discovery of tubercle bacilli in the feces. Acute ileocolitis has a more sudden onset and a shorter course, and the chronic form gives often the history of an earlier acute attack. Hemorrhage of considerable size suggests tuberculosis rather than ileocolitis.

Treatment.—This can be only symptomatic. Pain is to be relieved by hot applications to the abdomen, such as poultices or turpentine stupes, and if necessary by the internal administration of opiates. For the diarrhea bismuth and tannic acid preparations can be given, with or without opium. The diet must be sustaining but unirritating. Aleoholic stimulants are often required.

INTESTINAL HEMORRHAGE

As this symptom is referred to in various sections treating of special diseases it will be mentioned here but briefly. Strictly speaking intestinal hemorrhage indicates blood arising from the intestine itself, but more broadly the term may be used to include the discharge of blood from the rectum, whatever its source. Thus in severe epistaxis the blood may be swallowed and later passed from the bowel; and hemorrhage originating in the stomach may reveal itself in like manner. Melena is the title applied to one of the earliest forms of intestinal hemorrhage seen (see Melena, p. 266), the blood being usually altered in character and appearing as a tarry substance in the stools. In some cases this depends upon ulceration, especially of the duodenum. Another form of intestinal hemorrhage occurring very early in life is that observed in the hemorrhagic disease of the new born (p. 264), in which the loss of blood may be very considerable. Intestinal hemorrhage is also seen in the ulceration

of typhoid fever, intussusception and ileocolitis, the last two exhibiting usually streaks of blood merely. The hemorrhage of tuberculous ulceration may be of the same streak-like character or may be of considerable size and even large enough to be fatal. Hemorrhage from the bowel may occur in leukemia or pernicious anemia, is a common symptom of hemorrhagic purpura, and is sometimes seen in infantile scurvy. Bloody mucus or a few drops of blood with the stool are encountered in fissure of the anus and even of simple intense congestion of the intestinal mucous membrane, while ulceration of the rectum sometimes produces a considerable loss of blood, as may the hemorrhoids which occasionally occur in early life. Sometimes the hemorrhage depends upon the presence of a rectal polyp, or of small papillomatous growths. Apart from these conditions the appearance of blood in streaks, or even to the amount of a fluidram or more, frequently attends in infancy the injury to the rectal mucous membrane done by the passing of a large, hard fecal mass.

The treatment of intestinal hemorrhage depends entirely upon the cause and is referred to under the separate headings where these causes are discussed.

APPENDICITIS

This title has in recent years supplanted the older ones of typhlitis, perityphlitis, and the like, these conditions now being attributed invariably to a primary disease of the appendix. Although abscess in the cecal region had been recognized at a much earlier date, the first recorded case proven to have been disease of the appendix appears to have been that reported by Mestivier in 1759 (Deaver).¹

Etiology.—The disease may occur at any period, yet it is most frequent in early and middle life, being commonest between 10 and 30 years (Kelly and Herndon).² It is distinctly less frequent under 10 years of age. Hawkins³ in 224 cases of all ages found only 26 in children from 5 to 10 years. McCosh⁴ in 1000 operative cases of appendicitis at all ages recorded 17 in the first 5 years of life, 51 at from 5 to 10 years, and 85 at from 10 to 15 years; and Maguire⁵ in 104 collected cases in children found 3 under 3 years; 47 from 3 to 8 years; and 54 from 9 to 14 years. Writing in 1901,⁶ I was able to collect but 14 cases from medical literature occurring in the first 2 years of life, to which was added a 15th personal case in an infant of 3 months. Of the reported cases 2 (Pollard⁷ and Goyens⁸) were in infants of 6 weeks of age. Other scattered instances of the disease in infancy have been published since then, one by Remsen⁹ in an infant of 16 days, and Abt¹⁰ has collected in all 80 cases of the disease in the first 2 years of life. One of the youngest recorded cases appears to have been that of Gloniger (Kelly and Herndon)¹¹ in an infant operated upon successfully when but 41 hours old; while

¹ Treatise on Appendicitis, 1900, 18.

² The Vermiform Appendix, 1905, 452.

³ Dis. of the Vermiform Appendix, 1895, 62.

⁴ Journ. Amer. Med. Assoc., 1904, Sept. 24.

⁵ Virginia Med. Semi-month., 1898-99, III, 400.

⁶ Univ. of Pa. Med. Bull., 1901, Oct.

⁷ Lancet, 1895, I, 1114.

⁸ Gaz. méd. Belge, 1900, XII, 133.

⁹ Annals of Surg., 1912, LVI, 911.

¹⁰ Arch. of Ped., 1917, XXXIV, 641.

¹¹ *Loc. cit.*, 453.

Jackson¹ records an instance which he regards as prenatal, found in an infant dying of metallic poisoning when 40 hours old.

More males appear to be attacked than females, although some statistics are at variance on this point. In 500 cases in children up to 15 years seen by H. C. Deaver² there were 315 males and 185 females. Digestive disturbances, especially constipation or diarrhea, are perhaps the most frequent predisposing causes in children. Infectious diseases sometimes predispose. This is especially true of lacunar angina, although observed also in typhoid fever, grippe, rheumatism, and pneumonia. Trauma seems sometimes to be a cause, and heredity is also not without influence, there being a distinct tendency for more than one member of a family to be attacked. Although foreign bodies, such as fruit seeds, are sometimes, and fecal concretions often, found in the diseased appendix there seems little reason to believe that these have any etiological relationship except in occasional instances.

As to the direct *exciting* cause little positive is known, except that anything which produces narrowing of the lumen of the appendix may occasion retention of secretion, and be followed by congestion and by wandering of bacteria from the surface of the mucous membrane into the tissue of the wall of the organ. The germs found are most frequently streptococci and especially colon bacilli.

Pathological Anatomy.—The various divisions of appendicitis, based upon the pathological lesions and clinical symptoms, are, for the most part, only steps in the same anatomical process. As a result of, perhaps, kinking of the appendix or other cause obstructing its lumen, congestion takes place and inflammation of the mucous membrane follows with redness and swelling, especially of the lymphatic follicles which are extremely numerous in this organ, and which, in fact, make it resemble the tonsil to some extent. A small-celled infiltration accompanied by edema occurs which, with bacteria, may penetrate even to the serous layer. The appendix is thickened, stiff, and cylindrical and may be much distended by the secretion. A fibrinous inflammation of the serous layer may produce a thick deposit of fibrin on it and on the adjacent adherent coils of intestine. The condition produced is that denominated in its milder form *catarrhal appendicitis* or *appendicitis simplex*, or *diffuse appendicitis* when more severe. Entire resolution may take place, but often some degree of overgrowth remains, causing more or less constriction of the appendix, or the formation of adhesions. On the other hand, the process may advance further and may give rise either to a chronic inflammatory condition, or to suppuration. If bacteria have penetrated deeply and in large numbers, and the cellular infiltration and edema have been extensive, pus is produced in the wall of the appendix (*suppurative appendicitis*) and penetrates into its lumen, filling and distending this with an offensive, purulent material which may sometimes discharge itself through the natural opening into the bowel. More frequently, however, the pus makes its way also toward the serous layer and penetrates this (*perforative appendicitis*). This promptly produces a general septic peritonitis, unless the perforation is shut off by adhesions. In the latter event a localized peritonitis with perityphlitic abscess develops, which may finally find exit by eroding into the bowel or in other directions, but is especially prone to burst into the peritoneal cavity. In the more intense forms of suppurative inflammation, where the circu-

¹ Amer. Jour. Med. Sci., 1904, CXXVII, 710.

² Jour. Amer. Med. Assoc., 1910, LV, 2198.

lation is entirely cut off by the pressure of the exudate, there develops rapidly a gangrenous condition of all, or oftener of the tip, of the appendix (*gangrenous appendicitis*), which is followed by perityphlitic abscess if adhesions form, or frequently by a general peritonitis from rupture.

In cases of *chronic appendicitis* the appendix remains thickened and firm perhaps with constrictions at one or more portions, as a result of which the tip may be distended with pus or with a watery fluid, while numerous adhesions may connect it with other organs. A sudden severe acute attack may at any time develop upon the basis of the chronic disturbance. In other cases, especially infrequent in early life, as a result of repeated acute attacks a progressive involution of the appendix takes place, with atrophy of the lymph-nodes and the mucous glands, until the organ is much shrunken (*obliterative appendicitis*).

Symptoms.—These are sometimes very striking; sometimes recognized only with difficulty and uncertainty. In some instances they progress slowly and with no constitutional involvement; in others perforation with a septic peritonitis comes almost as out of a clear sky.

Catarrhal appendicitis may be so mild that it is not recognized at all and is supposed to be a mere digestive disturbance. In other cases the symptoms are more positive. In general this form of the disease develops as a primary affection or consecutive to some digestive disorder, and is ushered in by colicky pain in the right iliac fossa or elsewhere in the abdomen, this constituting the principal symptom. With this are often combined nausea, vomiting, moderate fever of 100° to 102° F. (37.8° to 38.9° C.), loss of appetite, coated tongue, and constipation or sometimes diarrhea. All these vary with the severity of the case. Examination reveals tenderness, increased resistance in the cecal region, and often in 1 or 2 days an induration which can be discovered by palpation. Often, too, when there is a plastic exudate upon the serous surface of the appendix and the neighboring parts, a distinct tumor can be palpated. If the patient is not confined to bed the manner of walking is suspicious, the child leaning forward and keeping the right thigh slightly flexed toward the abdomen. When in bed the patient lies on the back with the right thigh partially flexed.

Suppurative appendicitis is marked by the evident constitutional involvement, the rise of pulse-rate and perhaps of temperature, the appearance of the face, and other symptoms pointing toward a moderate degree of septic poisoning. These symptoms may develop with great rapidity after some days of manifestations of a mild character. In other cases suppurative appendicitis is present almost from the beginning, the onset is violent and acute, the fever more marked, vomiting troublesome, the pulse rapid and pain decided. These symptoms may continue for several days or they may subside promptly and lead to the conclusion that recovery is about to occur. Then in from 2 to 4 days from the onset perforation may take place.

The symptoms of suppurative appendicitis vary, whether with or without perforation. The inflammation may not pass beyond the serous wall of the appendix, and produce only moderate distension of the lumen of the organ, and no extensive induration develops. In those non-perforative cases in which a localized plastic peritonitis also occurs, a very decided tumor can be detected on palpation. Fever may continue or may subside, and the symptoms are less severe than in the cases in which a walled-in abscess forms about the appendix. In these latter vomiting tends to persist, tenderness and resistance are decided, pain is

variable but usually severe, and tympanites is common. The rapidity of the development of suppurative appendicitis varies greatly. In some cases, as stated, the course is rapid from the beginning and in 2 to 3 days undoubted abscess can be discovered. In others the early manifestations are all mild and the evidences of abscess develop only after a number of days and are vague. In still others the early symptoms may be severe and be followed by a period of comparative quiescence, lasting several days or even weeks and then the local and constitutional evidences of the formation of pus appear.

The constitutional symptoms of suppurative appendicitis depend not so much upon the local accumulation of pus as upon the degree of septic absorption which takes place. The temperature is subject to great variations in different cases and is not characteristic. Often the development of abscess is marked by a progressive increase of fever; while on the other hand the temperature may remain normal or nearly so even in cases which are clearly septic. The pulse becomes rapid and weak and the general sensations and appearance of illness increase in proportion to the degree of septic absorption occurring.

Perforation into the peritoneal cavity may take place from a gangrenous appendicitis which has formed no adhesions, or from a peri-appendicular abscess which has finally burst the restraining wall. It is characterized by the occurrence of vomiting, or an increase of this if already a symptom; severe abdominal pain; and profound collapse with the usual signs of rapid feeble pulse, shallow respiration, and fall of temperature. The expression of the face is anxious and pinched, cold perspiration occurs, the abdomen is extremely tympanitic, and the liver-dullness much diminished. Death may take place without any reaction, or the temperature may rise rapidly even to hyperpyrexia and the symptoms of septic peritonitis develop. The signs at this period may, however, be very deceptive, especially in children, there being sometimes only a moderate depression of temperature, with apparent improvement in the general symptoms attending the beginning of septic poisoning.

The symptoms of **gangrenous appendicitis** are very misleading from the beginning. The early ones are not characteristic and are often no more severe than those of catarrhal appendicitis. Suddenly, after a few days illness, perforation takes place with the symptoms of this as described. In other cases of gangrene the local manifestations are severe from the onset with unusual tenderness, pain and resistance of the abdominal walls.

Appendicitis in infancy exhibits symptoms which are liable to be very misleading, owing to the inability to determine with exactness the existence or position of pain and tenderness. Doubtless many cases are entirely overlooked at this age. The disease may exhibit a slow or sudden onset, troublesome vomiting, diarrhea or constipation, more or less fever, and finally peritonitis.

Recurrent and Chronic Appendicitis.—There is a very decided liability to the occurrence of repeated attacks of acute appendicitis. This depends, doubtless, on the persistence of kinking or narrowing of the tube, or on other causes which determined the first attack or which develop as a result of this. Among such causes may be the remaining of small, infected foci which at any time precipitate an acute inflammation. Such recurrences may finally lead to a severe and fatal appendicitis; or the disposition to them may at last disappear, perhaps through obliterative inflammation of the appendix. It is a mistake, however,

to assume that recurrences must necessarily come after the first attack. Hawkins¹ estimates the liability to recurrence as at least 23.6 per cent. as shown in the analysis of 250 patients of all ages. Fitz² found it in 11 per cent. in an earlier series of collected cases, but in 44 per cent. of 72 cases seen by him at a later period.

This condition of recurring attacks may be regarded as one of the forms of *chronic appendicitis*. In other instances the attacks are more frequent and so little marked that their nature is not recognized, and they are regarded as evidences of acute indigestion, until examination discovers the existence of an induration. In still other cases the disturbances are more of the nature of a chronic indigestion with poor or irregular appetite, constipation and other indefinite digestive symptoms, and general poor health. Pain may be very frequent and in some cases nearly constant; or may be brought on only by fatigue. It may be located in the appendicular region or be more diffuse. Some cases of repeated attacks of vomiting of the recurrent type probably owe their origin to actual appendicitis. The frequency of this has been maintained especially by Comby.³

In view of the importance of a prompt diagnosis of appendicitis and the great difficulty in recognizing the disease in many instances, a fuller consideration of the individual symptoms is of advantage.

Abdominal Pain.—This varies greatly in intensity; from severe suffering, either continuous or often occurring in paroxysms with intervals of nearly complete or entire comfort, to pain so slight that it is hardly noticed by the child or is more of the nature of an uncomfortable sensation. The mildest cases have but little pain and the severe ones suffer much from it; but this is true only to a limited extent, since serious cases sometimes have but little suffering even when abscess is forming or a short time before perforation occurs. The pain is often at first diffuse but later usually confines itself more to the right iliac fossa. To this there are many exceptions, and pain is referred to other regions of the abdomen, not infrequently the umbilicus or the epigastrium, and sometimes elsewhere. When it is in the region of the bladder and attended by symptoms of vesical irritability, as is often the case, it may readily lead to mistakes in diagnosis. Sometimes there is pain during or before evacuation of the bowels, and in other cases it is produced by traction upon the testicle or spermatic cord. On the occurrence of perforation there is usually a sudden, very severe, and more diffuse abdominal pain.

Tenderness.—This, like the pain, is far from uniform and depends to a certain extent upon the severity of the case, being but slight in catarrhal cases of the milder form. The occurrence of periappendicular inflammation is attended by increasing tenderness, and when abscess forms tenderness is very great. The situation of the sensation is usually in the right iliac fossa, most marked at McBurney's point, about midway between the umbilicus and the anterior-superior iliac spine; yet owing to the length of the appendix and its more variable position in children, the chief tenderness may be situated deeper in the pelvic cavity, and then is perhaps recognized only on rectal examination; or may be found higher in the abdomen than in adults or even in the left iliac region.

Increased Resistance of the Abdominal Walls.—This goes hand in hand

¹ *Loc. cit.*, 113.

² Boston Med. and Surg. Jour., 1890, CXXII, 619.

³ Arch. de méd. des enf., 1910, XIII, 401.

with tenderness and is very characteristic. It is nearly always present even in the mildest cases; and when tenderness is severe is so marked that a satisfactory examination cannot be made through the abdominal walls.

Induration or Tumor.—In the milder cases of catarrhal appendicitis there is no tumor, but only a thickened appendix which palpation may reveal. The rapidly gangrenous cases likewise may exhibit no tumor. On the other hand, tumor is present when the lumen of the appendix is distended by pus or when periappendicular inflammation occurs; whether this latter is purulent or merely plastic in nature. Bimanual palpation with one hand in the loin often aids in the examination. When tenderness is marked it is possible that the tumor can be detected only by rectal examination. This should always be practised in doubtful cases, comparing with the finger in the bowel the condition of the two iliac regions. Yet palpation of any sort must always be done with great gentleness, bearing in mind the danger of rupturing a periappendicular abscess. Percussion also is of aid in recognizing the tumor by the dullness of the sound produced. It should be done gently to avoid causing suffering. The size of the tumor varies greatly. In cases of purulent perityphlitis the abscess which forms sometimes reaches large dimensions. The position of the induration or abscess varies with that of the appendix. Like the pain, it is oftenest in the right iliac region, but may frequently be deeper in the pelvis, or occasionally below the liver or in the loin. The length of the appendix in children, and the fact that it is usually the tip of the organ which is the seat of the abscess-formation, occasion great variation in the position of the induration.

Tympanites.—This is not a pronounced sign in uncomplicated appendicitis. When decided it is an evidence of suppurative appendicitis, probably with a mild grade of peritonitis. When very great, with disappearance of hepatic dullness, it indicates perforation.

Nausea and Vomiting.—These are symptoms seldom entirely absent except in the very mild cases, but they are prone to subside after the first day or two. In the severer and rapidly developing attacks vomiting may be troublesome and persistent. Yet in many suppurative cases vomiting, like other acute symptoms, may lessen or disappear after the first few days, perhaps to return in force as the indication of the occurrence of a perforation. Stercoraceous vomiting is an evidence of absolute paralysis of peristalsis, such as is seen when widespread peritonitis develops.

Bowel Movements.—The condition of the stools is of importance in most cases. Diarrhea by no means excludes appendicitis in children, in whom it is more likely to occur than in adults. Generally, however, in severe cases, whether catarrhal or suppurative, there is more or less paralysis of peristalsis, and constipation is decided and sometimes absolute; and this is true of the instances of perforation also.

Temperature.—Fever is so extremely variable that few conclusions can be drawn from it. The mildest cases exhibit little or no elevation of temperature above a sub-febrile degree. Often there is some fever at the onset which may soon subside. In the severer attacks fever is liable to continue and reach a higher degree than in mild ones, although in septic cases the temperature may sometimes remain only slightly elevated. It is of frequent occurrence for the temperature to fall to nearly normal in patients who are, in fact, not improving, but developing abscess. A perforation may occur without premonitory return of fever; or renewed

rise of temperature may indicate a rapid abscess-formation. In fact, no reliable deductions can be based upon the elevation of temperature.

Blood.—The examination of the blood is sometimes a most valuable aid in the study of this disease. A rapid increase of leucocytes to 20,000 or 30,000 per c.mm., particularly of the polymorphonuclear cells, combined with the presence of other suggestive symptoms, is often an indication that the appendicitis is of a suppurative type. A diminution of a leucocytosis previously present is a favorable indication, if attended by an improvement in the general and local manifestations. On the other hand, little if any increase of the leucocytes is an equivocal sign. It may be present in catarrhal appendicitis or, conversely, in serious fulminating suppurative cases. The presence of a low leucocyte count with severe general symptoms is a bad indication. If the symptoms are severe, the higher the leucocyte-count and the greater the proportion of the polymorphonuclear cells the better is the prognosis.

Pulse.—The pulse at first is accelerated only in proportion to the degree of fever, or sometimes is slower than normal. If septic symptoms develop, and especially if perforation occurs, the pulse becomes rapid, weak and compressible.

Genito-urinary Symptoms.—These are sometimes decided. The urine is scanty and not infrequently contains albumin. Irritability of the bladder is sometimes great and may easily be a misleading symptom. An acute nephritis may develop.

Course and Prognosis.—The course of *catarrhal appendicitis* depends upon the severity of the inflammation. In the milder cases the fever, pain and vomiting disappear in 1 or 2 days, and at about the same time a slightly indurated appendix may be felt. The total duration is a week or less, although sometimes a longer interval elapses before the conditions disappear. In the more severe instances the initial fever and general symptoms last somewhat longer, and the indurated mass felt is very distinct; but improvement is not long delayed, and by the end of a week convalescence is well established, although induration and slight tenderness may last a somewhat greater time. If the inflammation does not advance to a suppurative condition the prognosis of the individual attack is always good, except for the great tendency to recurrence on one or several occasions, on any one of which a dangerous suppurative condition may develop.

Should the inflammation advance to a *suppurative stage* or be of this nature from the onset, the course varies, as pointed out in discussing symptoms. Periappendicitis may be produced and abscess result, and a mass may then often be outlined after 2 or 3 days, the general and other local signs meanwhile persisting. After this period the symptoms may continue with unabated force while the abscess grows larger; or they may abate after 4 or 5 days, except the local evidence of increasing induration. The duration of the attack in suppurative appendicitis has no well-defined limit. In some cases the course is rapid from the beginning and perforation may occur in 2 or 3 days, or suppuration be found if the case is operated upon. In others the abscess may grow slowly in size for several weeks. The ultimate termination in unoperated cases is very diverse. The pus, if in small amount, may be eventually absorbed, or it may extend itself in different directions and discharge spontaneously into the colon, the rectum, the bladder, through the abdominal wall, or by perforation into the peritoneal cavity. *Perforation* with general peritonitis occurs most frequently in cases of gangrenous appendicitis. This may

happen even in the first 2 or 3 days of the attack, with few unfavorable symptoms preceding the occurrence. The great majority of fatal cases of appendicitis depend upon perforation with general peritonitis. The prognosis in this condition is very grave, although cases of recovery under prompt operative treatment are not on the whole uncommon especially in infancy and childhood. The duration after perforation has occurred varies from 24 hours to a week or sometimes more, the longer period being the result of protective adhesions forming after a sudden perforation.

The general prognosis of appendicitis under *medical* treatment without operative interference is not unfavorable, owing to the large proportion of cases of catarrhal appendicitis. In fact, the large majority of unoperated cases in private practice recover. The prognosis of the individual case, however, is always uncertain, owing to the absolute impossibility of predicating the likelihood of peritonitis in any given instance. Those cases which perhaps appear the mildest or to be on the road to recovery not infrequently become suddenly and dangerously worse. The prognosis in the first 2 years of life appears to be unfavorable. This is probably dependent, in reality, upon the difficulty in recognizing the disease at this age, the mildest cases never being discovered, and the fatal ones receiving a post-mortem diagnosis only. Certainly the majority of patients at this age in whom the diagnosis is made during life do not recover. Recovery from an attack of appendicitis leaves the subject predisposed to later attacks, or chronic inflammation of varying degree may remain, or adhesions persist which give rise to symptoms.

Under early *operative* treatment the general mortality is very low. In the 500 cases in children reported by H. C. Deaver¹ there were but 23 deaths; *i.e.* 4.6 per cent., and the majority of these were due to general peritonitis. Riedel² found a higher mortality than this: 16.4 per cent. in 310 operated cases in children; but he thinks this high mortality due to neglect on the part of the parents, as a result of which operation was often done too late. One of the dangers after operation is the development of intestinal obstruction from paralysis of peristalsis; and the formation of secondary abscesses is another. Even cases with a primary walled-off abscess of considerable size generally recover when operated upon. It is only where general peritonitis has developed that the mortality of operation is high.

Complications.—Abscess in various parts of the abdominal and pelvic cavities has already been referred to. Pneumonia, hepatic abscess, phlebitis, and other evidences of sepsis may follow a septic peritonitis. Empyema may be the result of the penetration of a subphrenic abscess into the pleural cavity or of the sepsis following perforation. Pleurisy, not of a purulent nature, is a not infrequent complication. Appendicitis may be complicated by hernia, and the inflamed appendix may be found in the hernial sac. (See Hernia, p. 790.) So, too, appendicitis may be followed by intussusception, as in a child of 5 months reported by Rardin.³

Diagnosis.—The principal diagnostic symptoms in typical cases are sudden onset; early vomiting and fever; and abdominal pain, tenderness, increased resistance, and later induration or tumor especially in the right iliac region. But the variations as already described are so great that

¹ *Loc. cit.*

² Münch. med. Wochenschr., 1907, LIV, 2365.

³ Virginia Med. Semi-monthly, 1901, VI, 398.

diagnosis is often difficult, and the early distinguishing of the different forms of appendicitis from each other is practically impossible. The diagnosis in infancy is usually only conjectural, owing to the impossibility of obtaining answers to questions or of determining with any exactness the existence or position of pain.

A number of other morbid conditions are to be taken into consideration in reaching a conclusion. *Appendicular colic*, in which the contraction of the appendix in expelling retained secretion or fecal masses causes pain, is unproductive of fever, tenderness, leucocytosis, or the constitutional disturbance which appendicitis usually presents. When there is fever the diagnosis is at times uncertain. *Intestinal colic* may cause peculiar difficulty if there happens to be a large fecal accumulation in the colon, particularly the cecum. *Acute febrile indigestion* closely simulates many cases of appendicitis at the onset and diagnosis at first may be impossible. Generally, however, the pain is less intense and the constitutional symptoms less marked, except that the fever is often high. *Intussusception* might simulate appendicitis in the presence of constipation and of tumor. General symptoms, however, are absent early in the disease and appear only later; while appendicitis is ushered in by fever, vomiting, and other acute manifestations. *Ileocolitis* may resemble appendicitis and at first cause confusion through the early presence of vomiting and of abdominal pain; but the symptoms in general are so different that the diagnosis soon becomes clear. I have seen *acute tuberculous inflammation of the lymph-glands near the cecum* resemble appendicitis so closely that operation was performed with the mistaken diagnosis. Many similar cases are on record. The subject has been reviewed by Gage¹ and others. *Typhoid fever* may, at the onset, suggest appendicitis to a certain extent, through the vomiting and the tenderness in the right iliac region; but the course of the temperature, the absence of leucocytosis, and later the presence of the Widal reaction serve to differentiate. The diagnosis is, however, sometimes difficult and I have seen children with typhoid fever operated upon under the mistaken belief that appendicitis was present. The occasional reference by the patient of appendicular pain to the region of the right hip may cause the diagnosis of *hip-joint disease* to be made. Careful examination of the hip will prevent the mistake. *Ovarian disease* has likewise occasioned errors in several instances; and *urinary symptoms* may usher in appendicitis and cause confusion. The employment of a catheter, if there is retention, and the examination of urine obtained will aid in coming to a decision. Unusual location of the appendicular abscess leads to mistakes later in the disease, and the possibility of a *psoas abscess* from spinal caries simulating a perityphlitic abscess must not be forgotten. Inflammation of *Meckel's diverticulum* has repeatedly been supposed to be appendicitis, and operation has been done for this condition; and there exists no certain differential diagnostic feature. (See Diseases of Meckel's Diverticulum, p. 809.) The error of believing a *pleurisy* or *pneumonia* to be an appendicitis is probably much more frequent than ordinarily supposed, and has repeatedly led to operation upon perfectly normal appendices. Writing in 1903² I reviewed the subject with the report of a number of cases of what may be called "appendicular pneumonia," (see Vol. II, p. 80), and since then I have observed a number of additional instances and know of some where surgeons of experience have operated. It is of common occurrence,

¹ Boston Med. and Surg. Jour., 1915, CLXXIII, 301.

² Jour. Amer. Med. Assoc., 1903, Aug. 29.

especially in children, for the pain produced in the pleura to be referred through the intercostal and abdominal nerves to the region of the appendix. A mistake in diagnosis is to be avoided chiefly by careful examination of the lungs in every case of suspected appendicitis; by noting the increased rapidity of respiration in pneumonia; and by the fact that the abdominal resistance is generally relaxed during inspiration in this disease, but is maintained in appendicitis. Lastly it must not be forgotten that, in the case of older children especially, pain in the right iliac region may be *psychic* in nature, the result of the mental impression made by what the patients have heard regarding the disease. The absence of fever and of tumor is suggestive, although a simulated tenderness of course is present. The diagnosis is often difficult.

Treatment.—Non-surgical treatment is that indicated for catarrhal appendicitis, and the majority of such cases will recover. In view however, of the impossibility, already referred to, of determining whether a case is catarrhal, suppurative, or gangrenous, the only safe treatment is operative interference. This is especially true of infancy and childhood, at which time the danger of peritonitis is greater. If the patient is seen early operation should be done at once. If an exudate has already taken place, the time for operation is to be determined for each individual case, it being sometimes better to delay until the abscess has become more distinctly localized. Under this condition, if symptoms subside and pus does not form, operation may be unnecessary and resolution allowed to go on without interference. But here again we are confronted with the possibility of sudden perforation in cases where it was supposed that no suppuration existed.

When for any reason early operation cannot be done, or when the diagnosis is uncertain, and in cases seen later in which operation is deferred by surgical advice, medical treatment must be instituted. The patient must be kept at rest in bed, given a very light diet, and an ice-bag be applied over the seat of inflammation. Vomiting may need to be controlled by the temporary abstaining from food or the taking of it in very small amount, and the administration of appropriate remedies, such as the swallowing of ice, iced champagne, or the giving of lime water or bismuth. No saline or other purgative should be administered, and unirritating enemata should be employed if it is necessary to relieve constipation. If acute symptoms have subsided opium is better avoided if possible, as it obscures symptoms and increases constipation and tympanites. Cases where pain is very severe sometimes, however, make opium a necessity, and where operation cannot be done and the possibility of increasing tympanites by the drug is kept in mind, there seems no remedy more certainly indicated than opium for the relief of this symptom.

Where there have been several attacks of recurrent appendicitis, even though slight, it is best to remove the appendix, as there is no predicating under what inopportune circumstances a severe or even fatal attack might occur. The operation is then conveniently performed in the interval between attacks.

DISEASES CONNECTED WITH MECKEL'S DIVERTICULUM

The omphalo-mesenteric duct passing in fetal life from the ileum to the umbilical vesicle remains after birth in from 1 to 2 per cent. of all persons as what is known as "Meckel's diverticulum." This may exist only as a short patulous protrusion from the ileum, oftenest from its convex border and found at from 1 to 3 feet (31 to 91 cm.) above the cecum;

as a tube wholly or partially open; or as a cord merely, extending entirely or a portion of the way to the umbilicus, and in the latter event either free at its distal extremity, or attached to some other region, oftenest the mesentery. The organ is found much most frequently in males. Lesions are more likely to develop if the distal extremity is attached. As a rule the diverticulum gives rise to no symptoms whatever, yet it is oftener the cause of pathological conditions than is usually supposed. Series of cases of such disturbances have been reported, and I have reviewed the subject in a previous publication.¹ Only a brief résumé can be given here.

1. Strangulation of the Intestine by the Diverticulum.—This is the most frequent lesion found. Of Wellington's² 326 cases of disease of the diverticulum, 144 were instances of constriction of the intestine by this organ or its remains. In 991 cases of intestinal strangulation from different causes collected by Halsted³ 6 per cent. were dependent upon the diverticulum. The majority of these cases occurred after the period of childhood. The symptoms of strangulation by the diverticulum are those of intestinal obstruction in general, with the exception of intussusception, which has characteristic symptoms of its own.

2. Patulous Meckel's Diverticulum Opening at the Umbilicus.—This is an uncommon condition nearly always seen only in males, of which Strasser⁴ could collect but 63 instances. I have met with it in but 1 case. The mucous membrane of the diverticulum protrudes at the umbilicus, producing a small tumor covered by mucous membrane, and with a central fistulous opening. Should the entire wall of the intestine project as well as the mucous membrane of the duct, the tumor is larger and with two lateral openings. Strangulation of the projecting portion may occur. When the diverticulum is open throughout its extent, feces and even intestinal worms may be discharged at the umbilicus; but if closed at its proximal extremity, only mucus is passed at the opening. The cases of fecal fistula usually terminate fatally. (See also Diseases of the Umbilicus, p. 295.)

3. Invagination of the Diverticulum.—Of Wellington's cases 59 exhibited this lesion. Not only may the diverticulum be itself invaginated, but it may be followed by an intussusception of the ileum also. The accident takes place usually not before later childhood, this distinguishing it from ordinary intussusception, which is so much more common at a decidedly earlier age. The obstruction of the intestine is usually not complete, and the amount of blood discharged is small.

4. Volvulus of Meckel's diverticulum is a rare condition, either exceptionally of the diverticulum alone, or oftener of the ileum also secondarily to this, or dependent upon the presence of the diverticulum but without twisting of it.

5. Hernia of the diverticulum is occasionally seen. Gray⁵ collected 42 undoubted cases.

6. Diverticulitis.—The disease of the diverticulum bearing this title is of especial interest because it may simulate appendicitis so closely that the diagnosis is impossible. It is an infrequent condition, the largest series of collected cases reported being that of Forgue and

¹ Jour. Amer. Med. Assoc., 1914, LXII, 1624.

² Surg., Gynec. and Obstet., 1913, XVI, 74.

³ Annals of Surgery, 1902, XXXV, 471.

⁴ Med. Rec., 1903, LXIV, 933.

⁵ Brit. Med. Jour., 1907, II, 823; 1908, II, 909.

Riche¹ with 59 instances. About $\frac{1}{3}$ of the instances occur in children (Cahier).² The disease may be secondary to some other lesion of the diverticulum, or arise as a primary affection. The method of production and the pathological anatomy in this *primary* diverticulitis are entirely similar to those seen in appendicitis, and the different varieties are the same. The course may be acute or chronic. The symptoms strongly suggest appendicitis, and consist of abdominal pain, nausea, vomiting, often constipation, fever, leucocytosis, and the development of abdominal tenderness and resistance, with dullness on percussion. In a few cases discharge of blood from the bowel has occurred. In a case under my care severe anemia was produced in this way. Perforation and septic peritonitis may take place; or in other instances intestinal obstruction may result from compression by the inflammatory mass, or in other ways. In *secondary* diverticulitis intestinal obstruction may first occur, and later the evidences of inflammation of the diverticulum be added. The diagnosis of diverticulitis from appendicitis rests chiefly on the localization of pain to the right of the umbilicus and somewhat higher than McBurney's point, or in some more distant region. Further suggestive of diverticulitis are the absence or slight degree of tympanites, and in some cases the presence of blood in the stools. Nearly all cases, however, have been diagnosed as appendicitis.

The only treatment of diseases of Meckel's diverticulum is operative interference. Indeed, should the organ in a healthy state be discovered at any abdominal operation for other conditions, it is a wise course to remove it, since its presence is a constant menace to life.

CHAPTER VII

DISEASES OF THE INTESTINE (CONTINUED)

PROLAPSE OF THE RECTUM AND ANUS

Etiology.—This is a rather common affection of childhood, especially of the first 3 years of life, although not often seen in the first 6 months. It occurs oftenest where there has been repeated decided straining at stool, and hence it is a frequent complication of ileocolitis or of diarrhea from other cause. Chronic constipation may produce it for the same reason, as may stone in the bladder, phimosis or other urethral obstruction, thread-worms, rectal polypus, or other cause of tenesmus. It is also common in debilitated subjects in whom the sphincter ani has lost its tone, even without there having been excessive straining. Under these conditions it may be a complication of pertussis. The anatomical relationships of the rectum in early life favor the occurrence of prolapse, among them being its more vertical position and the less firm attachment to the neighboring parts. Sitting low when at stool and upon a vessel with a wide opening, as the ordinary chamber-pot, is another predisposing factor.

Symptoms.—The condition is practically an invagination developing at the anus. The prolapse may be only partial and limited to the opening of the bowel (*prolapse of the anus*), not more than a slight ring-

¹ Le Diverticule de Meckel, 1907.

² Rev. de chir., 1906, XXXIV, 338; 550.

shaped eversion of the mucous membrane showing itself. In the more typical, severer complete prolapse (*prolapse of the rectum*) there is a considerable portion of the rectum extruded including all the coats, and forming a sausage-shaped or more globular, soft, dark-red mass, somewhat furrowed, more or less coated with mucus, and often with slightly bleeding points especially if handled (Fig. 263). At the part furthest removed from the body is a small depression, indicating the much narrowed lumen of the gut. The presence of the tumor, if the case is an acute one, causes constant efforts at straining. As a rule the prolapse occurs only at stool, or when there is a straining effort from other causes; but sometimes, in severe and obstinate cases, even walking about the room may be sufficient to cause an extensive prolapse to occur, which may remain down with few subjective symptoms.



FIG. 263.—PROLAPSE OF THE ANUS.

From a patient in the Children's Hospital of Philadelphia.

Course and Prognosis.—In mild prolapse, as of the mucous membrane only, the condition is self-reducing after defecation, and examination shows only blood and mucus in small amount with the stool as an evidence of the disease. In more severe cases the compression of the extruded gut by the anal sphincter causes swelling and the prolapse persists unless treatment is employed to accomplish reduction. The prognosis is good, yet the duration may be long-continued before the tendency to recurrence is overcome. Rarely in some severe cases necrosis of the mucous membrane may result from incarceration.

Diagnosis.—This is usually easy. The only condition at all resembling it is an ileocecal intussusception which has reached the anus and is beginning to protrude from it. This may be recognized by the fact that the finger can be passed well upward between the sphincter and the protruding bowel.

Treatment.—The first indication is to replace the extruded bowel, should prolapse be present at the time. As a rule this is readily accomplished. The bowel and the hands of the physician are well oiled and a steady, gentle pressure made until normal relations are obtained. Should the oiled intestine slip too readily from the grasp of the fingers, a soft linen or similar cloth may be interposed. The central portion of the prolapse, showing the position of the opening, should be reduced first. Inverting the child may be useful in some cases. Sometimes the application of ice-cold compresses diminishes the congestion and swelling in

more obstinate cases and renders the reduction easier. It may be necessary to dilate the sphincter with the finger in cases where the prolapse has lasted some time.

The most important and most difficult part of treatment is to prevent recurrence. To accomplish this some support must be given, especially at the time of defecation, in such a way that the anal opening is narrowed. The child should lie upon its back or side, the feces being passed into a wad of oakum or other soft material, the nates being meanwhile pressed together by the attendant. Older children may sit upon a specially prepared seat which may be put in position over the ordinary infant's chair or the toilet (Fig. 264). The circular aperture is 3 or 4 inches (8 or 10 cm.) in diameter and the child is placed over this in such a way that the anus is immediately over the opening. The slot extending forward allows the passage of urine. The seat should be sufficiently high from the floor to prevent the thighs from flexing unduly on the abdomen, and the child should be prevented from leaning forward. The apparatus supports the tissues about the anus and prevents the spreading effect which attends straining efforts.

In cases where prolapse occurs very frequently, even without effort at stool, efficient support may often be rendered by drawing the buttocks firmly together with a broad strip of adhesive plaster crossing them transversely. This I have generally found effective. Constipation is to be prevented by gentle laxatives; diarrhea checked by appropriate remedies. Frequent bathing of the anal region with cold water helps to contract the anus and give tone to the parts. Sometimes the insertion of tannic-acid suppositories is of service in a similar manner. Tonic measures of various sorts are also required, such as effective hygiene, abundant suitable nourishment, and the administration of strychnine, quinine, or iron. The rectal injection of a solution of adrenalin chloride has been advocated, and good results claimed (Miscrocchi).¹ Should the measures recommended be ineffectual, operative procedures are required. These consist generally of cauterization of some sort, but so vigorous a treatment is seldom necessary. The injection of paraffin into the pararectal tissue has also been employed with success.

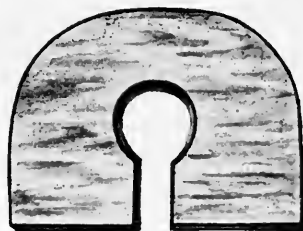


FIG. 264.—TOILET-SEAT FOR PROLAPSE OF THE RECTUM. To be used over the seat of the ordinary nursery-chair.

PROCTITIS

Inflammation of the rectum may attend ileocolitis, or, much less often, may occur independently of this. When the two are combined the symptoms are in no way characteristic, and need not be considered further here. Inflammation limited to the rectum will alone receive our attention.

Etiology.—Trauma is among the causes; such as is produced by too frequent administration of enemata, especially of an irritating nature. The use of the soap-stick or of glycerine-suppositories may occasionally act in the same way. Not uncommonly a gonorrhoeal inflammation of the rectum is produced by the extension from vulvovaginitis, or by the employment of an infected thermometer; or rarely in older children

¹ *La Pediatria*, 1905, XLII, 380.

by rectal copulation. The presence of the oxyuris may sometimes produce inflammation, and the disease may follow the infectious fevers.

Symptoms.—There is usually not the constitutional involvement seen in ileocolitis, the symptoms being mainly local, and consisting of rectal pain, tenesmus, and the discharge of bloody mucus. The latter is often not mingled with the stool, but perhaps passed before the feces appear. Irritation of the bladder not infrequently accompanies proctitis and prolapse of the rectum is common. When the inflammation is severe, ulcerative changes may take place in the rectum and the discharge contain pus, and not uncommonly blood in small or even large amount. In some instances of a more continued nature, as in those due to tuberculosis, ulceration may be present with but little pain, and there may be no tenesmus unless the lesion is in the lower part of the rectum. Occasionally a pseudomembranous inflammation of the rectum is found, depending upon the action of the diphtheria-bacillus or of the pneumococcus or streptococcus. The symptoms of this are those already described, with the addition of the discovery of the pseudomembrane by inspection of the bowel, or of the presence of it in the passages. The **course** of proctitis is usually acute, and the **prognosis** favorable, the symptoms subsiding in a few days. Sometimes, however, the condition passes into a chronic form.

Treatment.—The first indication is the removal of the cause. In the acute condition the patient should be kept in bed and given injections of boric acid, starch water or normal saline solution several times daily. The diet should be entirely unirritating and the tenesmus relieved by opiates if necessary. Later a weak solution of tannic acid (0.5 per cent.), or of silver nitrate (0.1 per cent.), or stronger solutions of argyrol or protargol are often of benefit. Suppositories of tannic acid may be serviceable.

FISSURE OF THE ANUS

Etiology.—Although not encountered so often as in adults, this is by no means uncommon in early life, being seen probably oftener in infancy than in childhood. Congenital syphilitic ulceration is an occasional cause, and injury done by the use of a syringe may occasion it. The irritation produced by the oxyuris or by eczema may result in fissure through the scratching which is indulged in. When occurring in older children, the most frequent cause is constipation with the passage of large scybalous masses. Once formed the fissure is likely to be kept open by the irritation of the feces passing over it, and by the alternate expansion and contraction of the anus which occurs during defecation.

Pathological Anatomy.—The lesion consists in a linear break in the mucous membrane discovered by careful examination at, or slightly above, the margin of the anus, and oftenest on the posterior wall. If of long duration it extends sometimes well above the anus, its edges are indurated, and the surface grey or yellow and secreting a small amount of pus, and bleeding slightly when touched. In the most severe cases there may be a number of such fissures present. To reveal the lesion the child should be laid upon the side or back with the legs flexed as closely upon the abdomen as possible, and the buttocks should then be pulled apart firmly, thus opening the sphincter and allowing the rectal mucous membrane to prolapse slightly. A digital examination of the rectum for polypus should also be made, as this condition sometimes accompanies fissure.

Symptoms.—The symptoms consist in pain on defecation and following it, and the discharge of a small amount of blood which may streak the stool. The pain is often very severe, and the patient abstains from defecation as long as possible. Sometimes incontinence or retention of urine is observed as a reflex disturbance, or pain may radiate to the legs, and may produce lameness and other symptoms strongly suggesting hip-joint disease. This has been emphasized especially by Svehla.¹ Various nervous symptoms occasionally develop.

Treatment.—That of the recent milder cases consists in keeping the stools soft, the rectum clean after defecation by the use of small enemata or of petrolatum on cotton, and the careful application of a 5 per cent. solution of nitrate of silver, or of the solid stick, followed by a soothing ointment. Sometimes the employment of a 2 per cent. cocaine-ointment, cautiously in young children, to the region of the fissure before defecation occurs anesthetizes the parts and prevents the pain. The passage of a stool may then be produced by a small unirritating injection, as of oil or of normal salt solution. In obstinate cases unrelieved by the procedures mentioned, dilatation of the external sphincter and cauterization of the fissure under general anesthesia may be required.

ISCHIORECTAL ABSCESS

Ischiorectal abscess is a by no means uncommon condition in early life, especially in infancy. It consists of an accumulation of pus in the cellular tissue about the rectum, and differs in no way from the condition as seen in adults. It may result from trauma, deep fissures of the anus, phlebitis of the hemorrhoidal veins, or infection of the lymph-channels. There develops fever without discoverable cause, since the abscess is often entirely overlooked for some time, owing to the impossibility of complaint by the youthful patient. Digital examination shows a bulging of the wall of the rectum, and inspection often an indurated or fluctuating red area in the anal region. Treatment consists in prompt, free incision. As a rule, the abscess heals readily, and only exceptionally does an anal fistula occur. Fistula is, in fact, of very rare occurrence in early life. According to Mitchener² in 1500 cases of this operated upon in St. Thomas's Hospital only 12 were in children.

INCONTINENCE OF FECES

For this disease there are various etiological factors. Lack of proper training may cause its persistence for some time after the control of the bowel should ordinarily have been attained. In these cases the trouble is but temporary. In greatly debilitated states and in the course of many severe acute diseases, fecal incontinence, similar to incontinence of urine, may be present for a time until convalescence from the primary disease is under way. In still other conditions incontinence of feces depends upon some local condition of the lower rectum and the sphincter, such as dysenteric diarrhea, or overdistention of the rectum from chronic constipation, or may be the result of an old prolapse of the rectum, a recto-vaginal fistula, or earlier stretching of the sphincter for fissure. The most serious cases are those associated with diseases of the mind and the nervous system. Thus well-marked cases of idiocy may never learn to exercise control over the bowels, and there may be incontinence in the

¹ *Jahrb. f. Kinderh.*, 1906, LXIII, 187.

² *Brit. Jour. of Surg.*, 1911 15, II, 361.

paroxysms of epilepsy or during severe chorea. Various lesions of the spinal cord may be attended by fecal incontinence, among them being spina bifida and the different forms of myelitis. For many of these cases there is no relief possible, while in others recovery may take place after a time, the prognosis depending upon that of the primary disease.

Exceptionally fecal incontinence is a purely functional disturbance analogous to enuresis. I have reported 1 such instance in a boy of 8 years,¹ and cases have been published by others, and the subject reviewed especially by Ostheimer.² In some instances incontinence of this nature has depended upon some reflex irritation; a vesical calculus in one of Ostheimer's cases, and apparently upon hypertrophy of the tonsils in an instance reported by Silvestri.³ Cases of this purely functional nervous nature seem much more common in boys. The incontinence may occur at night-time only, or in the waking hours as well; and the stools may be formed or loose in character. The disease may last from birth, or may come on only when the patient is deteriorated in health or is undergoing some special nervous strain. It may yield readily, or may prove troublesome to cure. General treatment is required, including that of a tonic nature, such as cool bathing, outdoor life, and the administration of strychnine or arsenic. Sometimes belladonna and the bromides act favorably, as in enuresis.

HEMORRHOIDS

The occurrence of hemorrhoids is very uncommon in childhood, although exceptionally they may be found very early in life. I have seen the disorder in an infant of 12 months. In rare instances it may be congenital (Milward);⁴ but the condition is then not in reality a pile, but a small benign neoplasm of a fibrous and fatty nature, and has no real resemblance to the ordinary hemorrhoid. As in adults, hemorrhoids may be either external or internal in nature, and are oftenest the result of chronic constipation. They may also be produced by sitting at stool upon a low wide-mouthed receptacle, as happens in the case of prolapse of the anus. Tonic remedies are usually indicated, since the children are frequently debilitated subjects. Other treatment is similar to that effectual in adult life; chiefly the employment of cold local bathing and a mild astringent ointment.

PRURITUS ANI

This annoying affection is not unusual in children, although less so than in adult life. It is a symptom of many diverse conditions. In children the most frequent cause is the presence of the oxyuris; but eczema, hemorrhoids, constipation, indigestion from improper foods, and slight prolapse may produce it likewise. The treatment is that of the cause, and this must be sought for diligently. As palliative measures, in cases where no certain cause is discoverable, application of cold water or of hot water is often serviceable, as are ointments of cocaine, tar, carbolic acid, menthol, and the painting with compound tincture of benzoin or with tincture of iodine.

¹ Arch. of Ped., 1899, June.

² Univ. of Pa. Med. Bull., 1905, XVII, 405; Jour. Amer. Med. Assoc., 1907, XLIX, 1115.

³ Gaz. degli. ospedali, 1904, XXV, 46.

⁴ Lancet, 1907, I, 1489.

FOREIGN BODIES IN THE INTESTINAL CANAL

The constant tendency of infants to put small objects into their mouths makes the swallowing of these a matter of great frequency; and even in older children the slipping of fruit-stones, pieces of bone, pins, and many other substances into the gullet and downward is not uncommon. The danger of these being arrested in the esophagus and stomach has already been referred to (pp. 695 and 717).

Symptoms.—In the intestine a foreign body causes, as a rule, little trouble. It advances steadily to the rectum and is expelled, its presence perhaps producing slight pain or abdominal discomfort or tenderness. This applies only to objects of moderate size, smooth, and of regular contour. Others may occasion severe pain and much intestinal irritation, and the passage of feces containing visible or occult blood; or they may even perforate the intestinal wall; or there may be obstinate vomiting if the article is of a size and nature to produce intestinal obstruction. Articles of small size sometimes become lodged in the appendix and may even be the cause of perforation here. As a rule, however, objects in the intestine, even if of irregular shape, travel on to the rectum and are passed in the natural way.

In the rectum foreign bodies produce no symptoms in most cases, but sometimes may be the occasion of pain there, or of tenesmus. Occasionally they enter the rectum through the anus, either by accident or having been inserted by the patient. This is, however, very unusual in children.

In the class of foreign bodies are to be included large *fecal concretions*, which may attain such a size when in the colon that they are readily felt through the abdominal wall and may be the cause of intestinal obstruction; or, when in the rectum, may be passed with great difficulty or only with extraneous aid. Small fecal concretions, greatly resembling date-stones in shape, are not infrequently found in the appendix, and are often mistaken for objects which have been swallowed.

Prognosis.—The prognosis of a foreign body in the intestinal canal is in general good. In cases where the body is large, or possesses sharp points or edges, the prognosis must be guarded until the object has passed the anus. If it enters the appendix it is unlikely to reënter the intestine. It is possible that in this situation it may cause no trouble, but it is in a degree a menace. In the intestine the body may remain for a few days to a week or occasionally decidedly longer.

Diagnosis.—This is to be based upon an unquestionable history of the swallowing of an object, and on the probability that it has left the stomach. A careful watch should be kept upon all the passages from the bowel; the stools being thoroughly shaken with water until soluble, and then strained in the search for the article. The use of the Röntgen ray is invaluable in the case of an object opaque to it, to determine the position of this and whether or not it is passing downward in a normal manner. Sometimes palpation of the abdominal wall may reveal the presence of a body if favorably situated for this investigation. This is especially true of fecal concretions.

As far as subjective symptoms are concerned, diagnosis is more uncertain. In children old enough to be influenced by suggestion, the alarm of and the questioning by the parents may readily elicit complaint of a purely hysterical nature, referred to various parts of the abdomen. If the trouble is in the rectum there may be tenesmus.

Treatment.—This must be expectant at first, and in emergency, surgical. The giving of starchy food, such as bread, arrowroot, oatmeal, and potato, tends to coat the body and render its passage through the intestine easier and less harmful. In the case of fecal concretions, repeated administration of purgative drugs in small dose may be needed, castor oil being one of the best of these. The occurrence of obstruction or of symptoms of peritonitis demands prompt operative interference, but this is seldom required. In the rectum the body may be sought by digital exploration and by the speculum, and its passage aided; if necessary by dilatation of the sphincter, although this will rarely be needed.

MORBID GROWTHS OF THE INTESTINE

Rectal Polypus.—Although not a common affection this is oftener seen in childhood than at other periods of life. It is, however, rare in infancy. The growth is an adenoma, pea-size or larger, of a bright-red color, and usually single and situated upon the posterior, or sometimes the anterior, wall of the rectum, 2 or 3 inches (5.1 to 7.6 cm.) above the anus. There is generally a pedicle $\frac{1}{2}$ to 3 inches (1.27 to 7.6 cm.) in length, but occasionally the growth may be attached to the mucous membrane by a broad base. Sometimes several polypi are present.

The chief *symptom* is hemorrhage, with or without the passage of mucus. If the pedicle is sufficiently long for the polypus to approach the anus there is also discomfort or pain in the rectum and tenesmus, and the tumor may be protruded through the anus during efforts at defecation. There may also be constipation or, occasionally diarrhea, and sometimes symptoms of nervous disturbance.

The *diagnosis* is to be made only from hemorrhage from other causes and from prolapse of the rectum; and careful examination readily shows the differences. *Treatment* consists in removing the growth by operative measures. A return of the trouble is unusual.

Other Morbid Growths.—These are of great rarity in early life. The most common is sarcoma, which may be either primary, or secondary to the disease in other regions. Nobecourt¹ was able to collect but 13 cases. Fibroma, angioma, lipoma and cysts have also been found. The lymphoid growths of leukemia may also occur in this locality; and Zuppinger² has collected 12 instances of carcinoma, including 1 reported by himself.

CHAPTER VIII

INTESTINAL PARASITES

The diagnosis of "worms," as made by the laity and frequently by physicians, is certainly oftener a mistaken than a correct one. Although still common enough, the frequency of the occurrence of intestinal worms has unquestionably diminished greatly. All sorts of symptoms are attributed to their supposed presence, but only the actual finding of the parasites or their ova justifies the diagnosis.

¹ *Traité des mal. de l'enfance*, 1904, II, 257.

² *Wien. klin. Wochenschr.*, 1900, XIII, 389.

ASCARIS LUMBRICOIDES

(Round Worm)

This nematode worm is perhaps the most common intestinal parasite in children in this country, with the single exception of the hook-worm as encountered in certain districts. From various statistics collected by Lechler¹ its occurrence would appear to be most frequent from 5 to 10 years of age. It is rarely seen in infancy. Perhaps the youngest recorded case was in an infant of 3 weeks, reported by Miller.² The total incidence of the disease is subject to wide variation with the geographical locality. Lechler's review shows a range of from 2.33 per cent. to 43.33 per cent. of the children examined. The ascarides occupy especially the small intestine, from which they may pass downward and be voided from the rectum, or they occasionally wander in other directions. Not infrequently they enter the stomach and may be vomited. They may also find their way into the larynx, causing asphyxia; the Eustachian tube; the nose; tonsils; the trachea and thence into the lung; the bile duct and thence the liver, where they have produced abscess; the pancreatic duct; the vermiform appendix, and Meckel's diverticulum, in the last perhaps being discharged from an umbilical fistula. Very rarely the worms may perforate ulcers in the intestine and enter the peritoneal cavity, causing peritonitis, and cases have been published in which even the healthy intestinal or gastric walls are said to have been completely penetrated. Plew³ has studied this occurrence, with the report of a case and a review of the literature. When in very large numbers they may occasionally produce intestinal obstruction. Of this rare occurrence Doberaner⁴ has collected 24 instances. (See Fig. 257.)

The round worm bears a close resemblance in form to the ordinary earth-worm, but is of a pinkish color and of larger size, being from $\frac{1}{8}$ to over $\frac{1}{4}$ of an inch (0.32 to 0.64 cm.) in thickness, and the male from 4 to 8 inches (10 to 20 cm.) and the female from 7 to 12 inches (18 to 31 cm.) in length (Fig. 265). The eggs, which are produced by the million, are round or oval, brownish or yellowish in color, about $\frac{1}{500}$ of an inch (0.005 cm.) in the greatest diameter and with a nodular outer coat. The number of worms ordinarily present in the intestine is usually not large, probably not exceeding from 5 to 20, but after a vermifuge they may occasionally be passed in handfuls and number even hundreds. The eggs enter the body through the mouth, having contaminated the drinking water or uncooked vegetables or fruits which have come in



FIG. 265. ASCARIS LUMBRICOIDES.
One-half natural size.

¹ Arch. f. Kinderh., 1913, XLII, 49.

² Jahrb. f. Kinderh., 1893, XXXVI, 319.

³ Arch. f. Kinderh., 1913, LXII, 11.

⁴ Prag. med. Wochenschr., 1914, XXXIX, 197.

contact with human feces, perhaps in the form of manure. They develop in the intestine and are mature in the course of a month. It is generally believed that no intermediate host is required, but that the eggs do not develop into embryos until they have been passed from the bowel and enter again by the mouth. Ransom and Foster¹ think no intermediate host is necessary, but that the worms as soon as hatched migrate to other organs, including the lungs, and thence enter the trachea, esophagus and finally the intestine, where they complete their development; while Stewart² maintains that rats and mice act as intermediate hosts.

Symptoms.—As a rule there are none whatever and the diagnosis can be made only by the discovery of the worms or their ova in the passages. Sometimes there are produced the symptoms characteristic of many digestive disturbances, such as irritability, restless sleep, grinding of the teeth, picking of the nose, colic, tympanites and loss of appetite; but these are much more frequently dependent upon other causes than upon the presence of the ascarides. Various nervous manifestations are often attributed to the parasites, and are sometimes actually due to them when they occur in large numbers, as proven by the fact that their removal by treatment may be followed by cessation of the disturbance. Among the assigned symptoms are attacks of fever, convulsions, choreiform movements, vertigo, headache, meningeal symptoms and anemia. It is possible that these are the result of the absorption of toxins produced by the parasite. An eosinophilia is generally present in the blood. The only serious symptoms are those seen in the rare instances referred to where a large mass of worms have occasioned intestinal obstruction; or where they have wandered into distant regions. The nature of these more remote disturbances depends, of course, upon the locality invaded.

Treatment.—This is usually very efficacious. The most popular and serviceable remedy is santonin in powdered form, combined with calomel or sugar, and given in doses of $\frac{1}{2}$ to 1 grain (0.032 to 0.065) to a child 4 or 5 years of age, 3 times a day, for 1 or perhaps 2 days. The patient should be prepared for the treatment by short starvation or the use of a milk-diet for a day or two, and a dose of castor oil or other purgative should be given after the course of santonin has been completed. When given with calomel, $\frac{1}{2}$ grain (0.032) of this to the dose, the castor oil will probably not be required. It is important to obtain purgation after the administration of santonin in order to remove it from the system, or it may be absorbed and xanthopsia, or "yellow vision" result, with headache, vomiting, vertigo and even convulsions. Another remedy sometimes employed is fluid extract of spigelia and senna, 1 fluidram (4) at 4 years of age, given 3 times a day for 2 or 3 days. Oil of chenopodium, 5 minims (0.31) on sugar at 4 years of age, administered 3 times a day, is also serviceable. Caution must be observed against an overdose. A couple of weeks after treatment with any vermifuge the stools should be examined for ova and the treatment repeated if these are found to be present.

OXYURIS VERMICULARIS (Thread-worm. Pin-worm)

The oxyuris is a nematode worm seen much most frequently in children, but is by no means confined to this age. I have found the worms in a man of 70 years who had suffered from them for an unknown period

¹ Journ. Agricult. Research, 1917, XL, 395.

² Parasitology, 1916-17, IX, 157.

in spite of repeated treatment for their removal. They are less often encountered in infants. A lack of cleanliness seems to predispose, but this does not appear to be essential. Trumpp¹ reported that in Munich 30 per cent. of the children examined showed the eggs or the worms themselves in the feces. The parasites inhabit the rectum and large intestine, the majority being found in the cecum; but they are not confined to this part of the gut, and in any event appear to enter it from the small intestine, which seems to be their breeding place. They are not infrequently discovered in the vermiform appendix and have occasionally been found in the stomach and even the mouth. Not rarely they wander into the vagina or under the prepuce, occur in the groins and about the genitals, and examination will generally reveal them in the folds about the anus.

In appearance the oxyuris resembles a very short white thread (Fig. 266). The female measures from $\frac{1}{4}$ to $\frac{1}{2}$ inch (0.64 to 1.27 cm.) in length and about $\frac{1}{25}$ inch (0.1 cm.) in thickness; the male not more than $\frac{1}{2}$ or $\frac{1}{3}$ of this size. In the intestines they are present in vast numbers, covering thickly the walls of the rectum, and being embedded in the mucus coating it. The number of worms passed is sometimes enormous. The eggs are produced in the bowel in large quantity, but are not always readily discovered in the stools. They are white in color, oval in shape but asymmetrical, with a smooth exterior, and measure approximately $\frac{1}{500}$ inch (0.005 cm.) in length and $\frac{1}{800}$ inch (0.0032 cm.) in diameter. The children constantly reinfect themselves and other children in the family by transmitting by the hand the eggs which have lodged under the finger nails in the act of scratching at the anus; or the eggs may come into contact with raw food, toys, dust, and the like. Thus introduced they develop into fully matured worms in the small intestine, and these may be found in the feces in 2 to 3 weeks. No intermediate host is required. It is generally believed that eggs produced in the intestine will not develop there until reintroduced by way of the mouth. This is, however, denied by Trumpp.



FIG. 266.—OXYURIS VERMICULARIS.
Natural size.

Symptoms.—The principal symptom is intolerable itching at the anus, usually worse at night-time and interfering greatly with sleep. Examination may reveal the living and moving worms in small or in great numbers coating the stools, or within the anus if the mucous membrane is slightly everted, or even in the folds of the groin or about the genitals. The giving of an enema will often bring away large numbers of the parasites from the bowel. The scratching which the itching incites often produces a secondary eczema about the anus. If the worms have entered the vagina vulvovaginitis results, and the intense itching may induce masturbation. Other secondary symptoms often appear, among them being enuresis, frequent micturition, prolapse of the rectum from straining, and catarrhal inflammation of the colon and rectum with discharge of a large amount of mucus. More remote symptoms sometimes result from the irritation and the loss of rest, such as night-terrors, anemia, debility, and even convulsions. Eosinophilia is an uncertain symptom, present in $\frac{1}{2}$ or less of the cases.

¹ Zeitschr. f. Kinderh., Orig., 1913, VI, 205.

Course and Prognosis.—The prognosis is good in many cases, when the worms are present in small numbers and the irritation slight. Here local measures may suffice. The great difficulty, however, which often renders successful treatment a puzzling problem, is the constant danger of reinfection when the worms are numerous and the itching severe. The impulse to scratch, which the child cannot resist and which takes place unconsciously at night, and the presence of numerous ova on the cutaneous surfaces in the neighborhood of the anus and even upon the nightdress and bed-clothing, render the transference to the mouth almost inevitable unless means are taken to prevent this. The fact, too, that local treatment by means of injection does not reach the upper colon satisfactorily or the small intestine at all, adds to the difficulty in curing severe cases, and renders the disease obstinate and the course prolonged.

Treatment.—This consists in (a) destroying by enemata in the rectum the female worms which are about to discharge their eggs; (b) the administration of remedies by the mouth which will kill the worms in the small intestine or upper colon; (c) the destruction of the eggs which have been deposited on the skin of the ano-genital region and, by the relief of itching and in other ways, the prevention of the carrying of them to the mouth of the patient with consequent reinfection.

(a) The first purpose is well served by the injection of an enema of infusion of quassia as high as possible into the colon with the hips elevated, using from a pint (473) to a quart (946) of liquid. This should be given every evening at bedtime for from 7 to 10 days. Success may be obtained also with large flushings with simple cold water or soap-water. Salt and water (1 ounce : 1 pint) (28 : 473), infusion of garlic, turpentine, vinegar, and corrosive sublimate (1 : 10,000) are also recommended, but those which are neither irritating nor poisonous in nature are to be preferred. For worms or eggs in the vagina the bichloride injection may well be employed.

(b) To reach the worms situated higher in the bowel various drugs have been recommended for administration by the mouth. Santonin, spigelia or chenopodium may be given as for ascarides. Naphthalene has been used by many, administering it in doses of $\frac{1}{2}$ to 1 gr. (0.032 to 0.065) 3 or 4 times a day at 2 years of age, and continuing for a week, the course being repeated after a period of 2 weeks. Oily substances should be withheld while this drug is being employed. Saline purgatives alone are excellent, citrate of magnesia often being taken readily by children. Any remedy employed is best given after a period of fasting.

(c) Preferably after every bowel movement, and certainly morning and night, the whole ano-genital region and surrounding parts should be bathed with a 1 : 10,000 bichloride solution. The necessity of the daily changing and thorough disinfection of the bedding and night-clothes is to be borne in mind. The hands of the nurse and of the patient must be kept disinfected; the dirt beneath the finger-nails where the eggs are so often embedded removed carefully; and at night, and possibly in the day also, the hands mechanically hindered from contact with the anus or the mouth, as by the wearing of mittens or other protective covering or of a pasteboard elbow-cuff which prevents flexion of the joint, or by dressing the child at night in close-fitting drawers. To relieve the itching and lessen the tendency to scratching, borie-acid ointment or mercurial ointment may be applied to the anus, especially at bedtime. This has the additional advantage of being destructive to the eggs and worms.

It must be remembered that the disease is very contagious and that other children, or even adults, of the family may be sufferers from it. To cure any patient it is therefore necessary that in the prevention of reinfection all sources of family-infection must be considered, and all those infected must be treated.

TÆNIA

(Tapeworm)

These cestode worms are of common occurrence in early life, their frequency probably equalling that in adults. In 9000 children in Denmark Schiodte¹ found tapeworm in 43. To this statement infancy offers a decided exception, tapeworms of most species occurring at this period only under unusual circumstances, perhaps dependent upon departure from the ordinary diet of milk. The youngest cases recorded in medical literature appear to be in 2 infants of 5 days (Müller)² and 4 days (Armor)³ respectively, but with our knowledge of the life-history of the parasite such an occurrence seems scarcely credible. *Tænia solium* is reported by Pardo⁴ in an infant of 5 months, and *tænia saginata* by Comby⁵ in one of 9 months.

The abode of the tapeworm is the small intestine, from which segments pass into the large intestine and are voided with the stools. The worm is of variable length, depending upon the species, and is composed of a series of flattened, white, opaque segments (*proglottides*) more or less rectangular in form and each sexually complete. The head (*scolex*) is the size of a small pinhead, and is followed by a thread-like neck, and this by the youngest segments, at first very small. These rapidly increase in size, until mature in from 3 to 3½ months, when those toward the lower end of the worm separate and are discharged from time to time, singly or in short series, new segments being produced by the head. The worm retains its position in the bowel by means of the hooks or the suckers with which the head is provided. The ripe segments, as passed, contain the eggs, which vary in size, shape and number with the species, and which exhibit the embryos within them on microscopic examination. The number of tapeworms present varies with the species. Sometimes 2 or even 3 different species of tapeworm may be found present simultaneously in the intestine, but this is unusual.

The tapeworm needs an intermediate host in order to reproduce itself, the species of animal depending upon the species of worm. The proglottides of the worm after passing from the intestine soften and the eggs are discharged, or in the case of *tænia solium* the eggs may be thus set free before they leave the intestine. These are then swallowed by some animal, penetrate its intestine, and develop in the muscles and other tissues into the larval form of the worm; in the case of the true *tæniidæ*, the *cysticercus*. The flesh of the animal after it is eaten by man sets free the larvæ in the intestine, where they develop into tapeworms. The eating of the flesh containing these is not, however, absolutely necessary, since drinking water contaminated by them may produce the disease. Such a method of infection is, however, rare. In the case of

¹ Hospitalstidende, 1902, X, 1211.

² Correspondenzbl. d. Württemberg. ärztl. Verein, 1837, VII, 80. Ref., Grimm, Münch. med. Wochenschr., 1914, LXI, 1780.

³ New York Med. Journ., 1871, XIV, 618.

⁴ Soc. Ginec. Espan. Ref., Grimm.

⁵ Archiv de méd. des enf., 1911, XIV, 525.

tænia elliptica infected insects are eaten unconsciously and the larvæ ingested in this way.

The tapeworms usually infecting human beings are of several varieties, the principal being *tænia solium*, *tænia saginata* or *mediocanellata*, *tænia elliptica* or *eucumerina*, *tænia nana*, and the *dibothriocephalus latus*.

Tænia Solium; Pork Tapeworm.—To this the name of *armed tapeworm* is also applied, from the double row of hooklets which surrounds the proboscis. There are also 4 sucking mouths. The adult worm measures 6 to 12 feet (183 to 365 cm.) in length, the segments averaging about $\frac{1}{2}$ inch (1.27 cm.) in length and $\frac{1}{4}$ to $\frac{1}{3}$ inch (0.64 to 0.85 cm.) in breadth. The ripe segments toward the end of the worm are longer and narrower. The proglottides contain in their interior the dendritic uterus with 8 to 12 rather thick lateral branches on each side. The eggs are brown, spherical, and about $\frac{1}{800}$ inch (0.0032 cm.) in diameter. This tapeworm is acquired by eating the raw or imperfectly cooked "measly" flesh of the hog. Owing to the peculiarity of the *tænia solium*, that the eggs are often freed from the proglottides in the intestine and passed by stool, patients may occasionally, through uncleanly habits, reinfect themselves by swallowing the eggs and in this way develop the cysticercus in various parts of the body. *Tænia solium* is very much less common in this country than is the *tænia saginata*, smaller in size, and is quite infrequent in children. It occurs almost always only singly in the intestine.

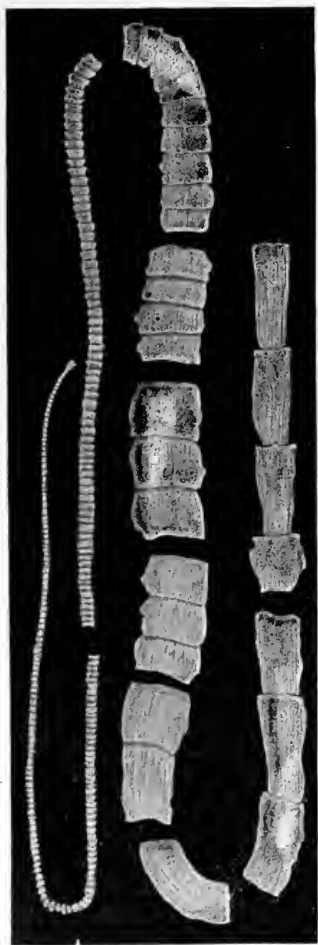


FIG. 267.—*TENIA SAGINATA*.
Different portions of worm.
Natural size.

Tænia Saginata; Tænia Mediocanellata; Beef Tapeworm (Fig. 267).—The head is larger than in the preceding species, pear-shaped, with 4 sucking mouths, but not provided with hooklets or a beak. This tapeworm measures 15 to 24 feet (456 to 730 cm.) or more in length, $\frac{3}{5}$ to $\frac{4}{5}$ of an inch (1.5 to 2 cm.) in length and about $\frac{1}{2}$ inch (0.85 cm.) in breadth. Toward the middle of the worm the breadth is greater than at other parts. The uterus contains 20 to 25 slender branches on each side, and the eggs are oval and measure $\frac{1}{800}$ to $\frac{1}{600}$ of an inch (0.0032 to 0.004 cm.) in length by somewhat more than half this in breadth.

The worm is acquired by eating the affected "measly" flesh of cattle which have themselves become diseased by ingesting the eggs of tapeworms upon vegetation which had come in contact with contaminated human manure. One or several worms may be present in the intestine at the same time. This species is very widely distributed geographically, and, although less common in the United States than in many other regions, it is by far

the most frequent variety found. It is rare in infants, but is occasionally acquired through the giving of scraped raw meat. I have observed the disease developing from this cause in several instances under 2 years of age.

Tænia Elliptica or Cucumerina; Dog Tapeworm (*Dipylidium caninum*).—This is a small tapeworm common in the dog and cat, but rare in the human race. Zschokke¹ collected 36 published cases, to which he added another; and Lins² raised the total number of reported cases to 68. The majority of instances of tapeworm of this variety have been observed in children and infants. Usually the parasite occurs singly, but Lins found from 20 up to 200 present at a time. The second host is the louse and the flea infecting dogs and cats, as well as the human flea. The close contact which play often brings with these domestic animals, and the natural tendency of the infants to put their hands to their mouths, allows of the swallowing of the infected insect and the subsequent later development of the tapeworm. This worm is slender and measures only 4 to 12 inches (10 to 31 cm.) in length. The head is armed with hooklets and a beak. The proglottides are from $\frac{1}{3}$ to $\frac{1}{2}$ inch (0.85 to 1.27 cm.) long and about $\frac{1}{3}$ as broad. The eggs are about $\frac{1}{500}$ inch (0.005 cm.) in diameter, from 6 to 12 being contained in a common capsule.

Tænia Flavopunctata; Rat Tapeworm (*Hymenolepis flaropunctata*).—This parasite has been rarely seen in infants and children. It is common in the rat and mouse.

Tænia Nana; Dwarf Tapeworm (*Hymenolepis nana*) (Fig. 268).—This is another variety of tapeworm which has been thought to be rare in this country, although more common in Italy. Ransom³ collected in 1904, 106 published cases occurring in man. The parasite is probably much more frequent than these figures represent, inasmuch as Amesse⁴ counted 68 cases observed and reported in America; Greil⁵ discovered it in 5.75 per cent. of 665 children in Alabama, ascarides being found in but 4.06 per cent.; and Schloss⁶ found it present in 14 out of 230 children examined (6.08 per cent.). Its most frequent habitat is the small intestine of species of rats and mice. The parasite is only $\frac{1}{2}$ to 1 inch (1.27 to 2.54 cm.) long, and has a spherical armed head, and 150 or more short and broad proglottides each containing 80 to 100 eggs. It occurs chiefly in children and often in very large numbers in a single case. Even nurslings may be affected. The intermediate host is unknown, and it is possible that none exists, but that the larva occupy the mucous membrane of the intestine and there develop into the perfect worm.



FIG. 268.—HYMENOLEPIS NANA.

A, One-half actual size; B, enlarged; showing head and suckers. (Schloss, Arch. of Pediat., 1910, XXVII, 1915.)

¹ Centralbl. f. Bact. u. Parasitenk., Orig., 1905, XXXVIII, 534.

² Wien. Klin. Woch., 1911, XXIV, 1565.

³ Public Health and Marine Hosp. Service, U. S. Hygiene Lab., 1904, Bull. No. 48.

⁴ Colorado Med., 1910, VII, 143.

⁵ Amer. Jour. Dis. Child., 1915, X, 363.

⁶ Arch. of Ped., 1910, XXVII, 100.

Dibothriocephalus Latus; Fish Tapeworm.—The head of this parasite is small, wedge-shaped, grooved on each side, and unarmed with beak or hooklets. The proglottides are broader than long, measuring $\frac{1}{2}$ to $\frac{3}{5}$ inch (1.27 to 1.52 cm.) in breadth and but about $\frac{1}{5}$ inch (0.51 cm.) in length. This feature easily distinguishes the parasite from other tapeworms. The sexual openings are on the surface of the proglottides instead of at the edge as in other varieties, and the uterus is rosette-shaped, instead of branched. The eggs are about $\frac{1}{400}$ inch (0.006 cm.) long and $\frac{1}{600}$ inch (0.004 cm.) broad, and are characterized by a lid-shaped closure at one end. After leaving the intestine the ova develop in water into a free-swimming infusorial organism, and then by way of the intestine enter the muscles of certain species of fish, where they remain as unencysted elongated larva of perhaps several inches in length, suggesting the appearance of the fully developed worm. The eating of fish thus infected produces the sexually active parasite in the intestine of man. The worm is 25 to 30 feet (762 to 915 cm.) or more in length. It is common in certain regions, as Switzerland and Scandinavia, but rare in the United States. Edsall¹ could find up to 1904 but 22 cases reported in America. I have, however, seen it once in the adult and again in a girl of 9 years, both unpublished cases.

Symptoms of Tapeworm.—As a rule, tapeworm produces no symptoms other than the passing of segments of the worm in the stools, and this constitutes the only positive diagnostic evidence. An eosinophilia is witnessed in rather less than half of the cases. In a small proportion there are various digestive disturbances present, such as occasional vomiting, abdominal pain or discomfort, excessive appetite, diarrhea, vertigo, headache, and the like; but in the majority of these it is doubtful whether the worm possesses any etiological relationship. This is equally true of the more distant reflex symptoms which have repeatedly been described, such as epileptiform and choreiform conditions. The only exception is the severe anemia of the pernicious type which sometimes clearly depends upon the presence of the *dibothriocephalus*.

Course and Prognosis.—It is uncertain how long the presence of the worm may continue. Certainly this may even be for years, new segments being produced as the mature ones separate, the total length of the parasite being thus unaltered. The danger to the patient is insignificant, barring the possibility of anemia or nervous symptoms referred to, and the chance of reinfection and the production of cysticercus in the case of the presence of *tænia solium*. The prognosis of cases subjected to treatment is on the whole good, although repeated efforts lasting over months are sometimes required before final relief is obtained.

Treatment.—In the way of prophylaxis care must be taken to prevent infection. All meat eaten should be sufficiently well-cooked to kill any larvæ present. In the days when the giving of scraped raw meat to children was common, the development of the beef tapeworm could occur more readily. Careful, frequent disinfection of the hands of little children and the avoidance of too close contact with dogs and cats are prophylactic measures against *tænia elliptica*.

For the actual treatment of the disease some vermifuge is required, but only after a positive diagnosis is made. Whatever this may be, to make the treatment effective the head of the parasite must be expelled. It often happens that although no head can be found, later experience with the case shows that it clearly had been broken off and passed, but

¹ Amer. Med., 1904, Dec., 1087

was undiscovered. Should the head have remained in the intestine but the rest of the worm have been discharged, the elapse of about 3 months' time is required for the maturing of segments which would be revealed again in the stools.

The treatment should follow a certain routine. The child should receive a light dinner and still lighter supper, such as a bowl of broth or of bread and milk. On waking in the morning, an enema and a saline purgative should be given. The object of this is to empty the intestinal canal as thoroughly as possible. Breakfast should be withheld, or consist of a cup of clear broth or beef tea. After the action of the purgative, the vermifuge is administered in a single dose or divided doses, and followed in an hour by castor oil or a saline in sufficient amount to insure thorough emptying of the bowels. When the desire for an evacuation comes the child should be seated on a full vessel of water so that the nates are in contact with the fluid. In this way the weight of the worm is partially supported and the danger of the breaking off of the head is less. No traction whatever should be made upon it but, if it ceases to pass easily, the nozzle of a syringe may be very carefully inserted into the anus and an enema given. After the passing of the stool and the parasite into the vessel of water, this latter should be rocked or stirred gently in order not to break the worm, and the fecal matter poured off; then more water added and the process thus continued until the worm is clean, after which, still suspended in water, it can be examined with care for the head.

As the remedies for tapeworm are often nauseating, the child should be kept reclining in bed after taking them. So, too, as the treatment is rather exhausting, further confinement to bed for a day or two is advisable, combined with the giving of a light diet, principally milk. If it is evident from the number and character of the segments that the worm is not completely removed, another effort should be made after an interval of some weeks.

The number of vermifuges recommended is large. All of them are supposed to have the property of killing, or at least numbing, the parasite and thus releasing the hold of its head on the intestine. One of the most popular and effective is male fern (*felix mas*), of which the oleoresin or the freshly prepared 'etheral extract may be given in doses of $\frac{1}{2}$ to 1 fl. dram (1.8 to 3.7) to a child 4 or 5 years of age. This may be made up into an emulsion disguised as far as possible; or in the case of older children given in capsules holding 15 minims (0.92) and administered every half hour until the complete amount is taken. In some troublesome cases success has followed the employment of a very light diet for a day or two, combined with the repeated administration of turpentine; then finally the giving of the male fern in the manner described.

Another very useful remedy is pomegranate (*granatum*), which may be given in decoction in doses of $\frac{3}{4}$ to 1 fl. oz. (22.2 to 30) for a child of from 5 to 10 years of age. As it is, however, of an unpleasant taste and liable to cause vomiting, the use of the alkaloid pelletierine is to be preferred. I have had good results with a preparation of this (Tanret's) in some obstinate cases where male fern had failed. The drug is liable to produce vertigo, fainting and nausea, and the child should be kept at rest, as already advised whatever tænicide is chosen. Kousso is often an effective, but always a very disagreeable remedy and one liable to cause vomiting. Pumpkin-seed is safe and not unpleasant to take, but is very uncertain in its effects. Its condensed extract is highly praised by Langer¹ and others.

¹ Pfaunder and Schlossmann, *Handb. d. Kinderh.*, 1906, II, 1, 230

Treatment for tapeworm should not be given to delicate children, or those suffering from digestive or other disturbances of moment, until the maladies have been relieved and the general health improved.

UNCINARIA

(Hook-worm. *Ankylostoma*)

Various species of uncinaria are known to infect animals, but only two have the intestine of man as their habitat. The first of these is the uncinaria duodenalis (*anchylostoma duodenalis*), first described by Dubini in 1838.¹ Since then this worm has been recognized as the cause

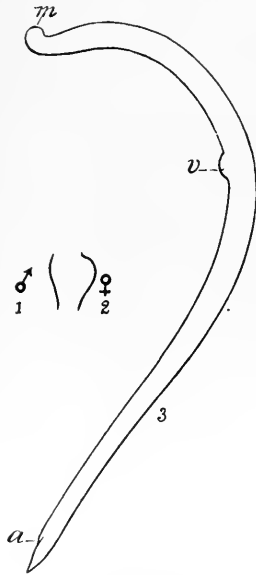


FIG. 269.

FIG. 269.—NEW-WORLD HOOKWORM. (*Uncinaria Americana*.)

Natural size: 1. Male; 2, female; 3, the same enlarged to show the position of the anus, *a*; the vulva, *v*; and the mouth, *m*. (Stiles, 18th Ann. Report Bureau of Animal Industry, 1901, 190.)

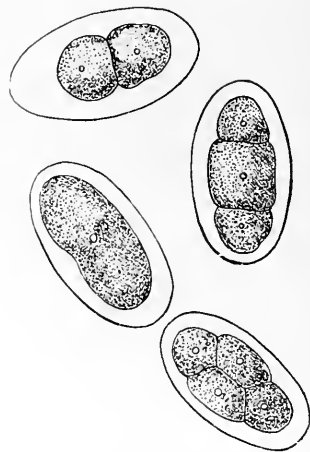


FIG. 270.

FIG. 270.—FOUR EGGS OF THE NEW-WORLD HOOKWORM.

Eggs exhibiting in the one-, two-, and four-cell stages. The egg showing three cells is a lateral view of a four-cell stage. Greatly enlarged. (Stiles, 18th Ann. Report Bureau of Animal Industry, 1901, 193.)

of certain forms of anemia, seen especially in those working much in earth. It is very widespread in tropical and subtropical countries of the Old World, although much most frequent in certain regions, abounding, for instance, in Egypt. In the United States it would seem not to be of common occurrence, only about 35 cases having been recorded by Stiles up to 1902.² Ashford³ was the first to point out the seriousness of ankylostomiasis in this country, finding it extremely common in Porto Rico; but Stiles in May, 1902,⁴ reported the discovery that the hook-worm of the

¹ Annali universali di medicina, Milan, 1843, CVI, 5.

² Bull. No. 10 Hygienic Lab. U. S., 1903, 9.

³ New York Med. Jour., 1900, LXXI, 552.

⁴ Amer. Med., 1902, III, 777.

United States was a different species from that seen in Europe, and named it the *Uncinaria Americana*; and studies of recent years have shown that uncinariasis is one of the most frequent, widespread and important diseases of the Southern States. It prevails especially in rural districts where the soil is sandy, and it appears most common and serious in women and children. Greil¹ found it in 26.75 per cent. of 665 children under 12 years, 80 of whom were Negroes. Although it affects especially the poor and dirty, it is by no means confined to these. Thus Gage and Bass² examined 315 students at Tulane University, New Orleans, and found 25 per cent. infected with hook-worm.

Uncinaria Americana is a very small, thread-like nematode worm, the female being about $\frac{1}{2}$ inch (1.27 cm.) in length and the male slightly smaller (Fig. 269). Its buccal orifice is provided with a pair of cutting



FIG. 271.—PENETRATION OF THE SKIN BY UNCINARIA.

Some of the parasites are seen already beneath the surface. (Ferrell, *Jour. Am. Med. Assoc.*, 1914, LXII, 1937.)

plates. The numerous thin-shelled eggs are elliptical in shape and measure about $\frac{1}{400}$ to $\frac{1}{350}$ of an inch (0.006 to 0.007 cm.) in length and about $\frac{1}{2}$ as much in breadth (Fig. 270). It is characteristic of them that they are usually more or less segmented. The *uncinaria duodenalis*, or Old World hook-worm, differs in being slightly longer and stouter, and in having the buccal orifice armed with teeth. The eggs are slightly smaller.

The life history of the *uncinaria duodenalis* is known, and that of the *uncinaria Americana* is probably the same. The worm inhabits the small intestine, especially the duodenum and the jejunum, and may be present in enormous numbers. By its sucking apparatus it attaches itself to the intestinal mucous membrane and probably abstracts blood from it. The head, by turning backward, gives the parasite the form

¹ *Amer. Jour. Dis. Child.*, 1915, X, 363.

² *Arch. Int. Med.*, 1910, VI, 303.

of a hook. In shifting its position the worm leaves minute bleeding points. It is likely, too, that it produces a poisonous substance.

The eggs will not mature within the intestine, but are passed with the feces, and enter moist ground or water where they promptly develop into the worm-like larvæ. These remain alive perhaps for months until they again enter the alimentary canal, being conveyed by the hands soiled by earth, or through the swallowing of contaminated water or the direct eating of earth according to the habit of some of the natives (*Geophagi*). In the intestine they finish their transformation, if sufficiently well-advanced, into the adult parasite. The larvæ may also reach the system by penetrating the skin directly from without (Fig. 271). Thence by way of the circulation they reach the pulmonary alveoli. From here they migrate along the bronchial tubes and trachea to the gullet, and thence to the esophagus and finally into the stomach and intestine (Looss).¹ No intermediate host is therefore needed. The worms very probably live in the intestine for years. A period of incubation of from 4 to 6 weeks is required from the time of the entrance of the larvæ before maturity is reached.

Symptoms.—It is likely that a large number of parasites must be present to produce symptoms. After an initial gastrointestinal disturbance, evidences of disease are entirely of a constitutional nature, and consist chiefly in the varied manifestations of anemia, this resulting from the direct loss of blood, from the poison entering the system from the mouth of the worm, or through the entrance of bacteria through the wounds. The intensity of the symptoms varies with the case. In well-marked instances the complexion grows pale and clay-colored, the expression is apathetic and of a peculiar dullness; the abdomen is much distended by gas and sometimes contains fluid, the liver and spleen are enlarged, the growth of the body is stunted, emaciation is present, and there may be edema, especially of the face. Pica, or the habit of eating dirt and the like, is a common symptom, probably oftener the result than the cause of the disease. There may be subnormal or sometimes elevated temperature. The blood exhibits marked reduction of the red blood-corpuscles and especially of hemoglobin, often to 20 or 30 per cent., while an eosinophilia, commonly of 8 or 10 per cent. or more, is a very constant and characteristic symptom.

Prognosis.—The prognosis for recovery from the disease is unfavorable unless treatment is instituted. This is especially true of children. Yet, although many deaths occur, there is even greater disposition for the disease to become chronic and to last for years until terminated by some complicating affection. Many adults show a tendency to recovery, if reinfection does not occur. Under treatment the prognosis is favorable, except in the advanced cases with great debility and anemia.

Diagnosis.—If the disease is suspected, and examination of the feces made, the diagnosis is easy, especially if treatment has been recently given and a large number of eggs are passed. The segmented appearance of the eggs is quite characteristic. The presence of eosinophilia is also suggestive. It is important to bear in mind that a large proportion of the cases supposed to be chronic malaria, anemia, and the like, are in reality uncinariasis.

Treatment.—**Prophylaxis** consists in hygienic precautions against infection in the districts where the disease prevails; these consisting

¹ Centralbl. f. Bakt. und Parasit., 1898, XXIV, 484.

especially in the frequent washing and disinfection of the hands, the disinfection of the feces, the boiling of drinking water, and the forbidding of the children to run barefooted. In regions where the disease is endemic, the building, correct location, employment, and disinfection of properly constructed privies should be insisted upon, and the disinfection of the ground about the dwelling-houses accomplished by the application of fire in some way.

Direct treatment is usually simple and efficacious. After a very light diet for 24 hours, thymol may be administered to a total amount of $\frac{1}{2}$ fl. dram (1.8) in divided doses to a child of 10 or 12 years, followed in 2 hours by a saline. No castor oil or other fatty substance should be given during the treatment. The drug may be made into an emulsion or put into capsules. The feces should be examined after a week to see that no more eggs are present. If they are still found, the treatment must be repeated, or *felix mas* used in the manner recommended for tapeworm. In weakly subjects the thymol should be administered in smaller and more frequent doses, since the drug is capable of producing dangerous symptoms. Oil of chenopodium is recommended in place of thymol by Levy,¹ Bishop and Brosius² and others as more efficacious and safer. The dose is 1 drop (0.062) on sugar every 2 hours for 3 or 4 doses for each year of the child's age. The last dose should be followed in about 2 hours by castor oil. A saline laxative should be given on the day preceding the administration of the vermifuge. Hall and Foster³ recommend chloroform as far superior to chenopodium. The adult dose is 2 to 3 c.c. (33 to 49 m.) dissolved in castor oil. After the removal of the parasites tonic treatment directed to the anemia is required.

OTHER INTESTINAL PARASITES

Other animal parasites occurring in the intestine are of minor importance. Chief of them is the *trichocephalus dispar* or **whipworm**, a small slender nematode worm about $1\frac{1}{2}$ to 2 inches (3.81 to 5.08 cm.) long, for two-thirds of its length very thin and thread-like, and then joined like a whip-lash to the thicker posterior portion, which in the male is rolled up like a spring (Fig. 272). The numerous eggs are lemon-shaped and of about the size of those of the oxyuris— $\frac{1}{500}$ inch (0.005 cm.) in length and $\frac{1}{800}$ inch (0.0032 cm.) broad—but with a plug-like closure at each end. The parasite, seldom in large numbers, inhabits especially the cecum. It is of very common occurrence in some countries, less so in the United States; although Townsend⁴ discovered it in the feces of

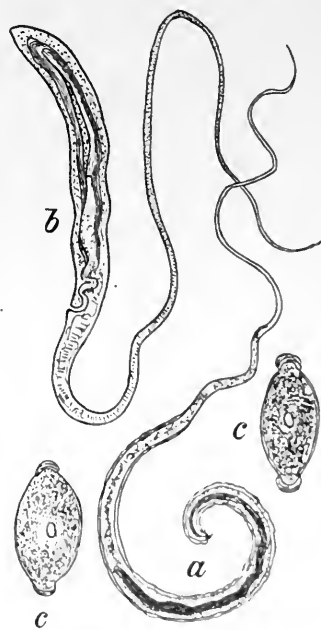


FIG. 272.—*TRICHOCEPHALUS DISPAR*.

a, Male; b, female; c, eggs.
(von Jaksch, *Klinische Diagnostik*, 1887, 150, Fig. 54.)

¹ Journ. Amer. Med. Assoc., 1911, LXIII, 1946.

² Journ. Amer. Med. Assoc., 1915, LXV, 1610.

³ Journ. Amer. Med. Assoc., 1917, LXVIII, 1961.

⁴ Amer. Text-Book of Diseases of Children, 1891, 553.

all of 16 children examined. Neumann¹ found the parasite in the feces of 13.9 per cent. of 122 children of from 1 to 16 years, and it is perhaps as often seen in adults. Christoffersen² in 200 autopsies of various diseases at all ages observed it in 29 per cent. It is often associated with other worms, especially the ascaris. The ova develop in water or damp earth, and are thence taken by accident into the stomach. There are practically no symptoms, except it may be diarrhea or anemia as reported in some instances. Treatment is unsatisfactory, the worms seeming to be almost impossible to remove by the use of drugs. Various **protozoa** and **infusoria** are from time to time found in the intestinal canal in children, among them the *Ameba coli*, the *Cercomonas intestinalis*, the *Trichomonas intestinalis*, the *Balantidium coli*, and the *Megastomum entericum*. They are recognized by microscopical examination of the stools, but for details of their appearance reference must be made to works especially devoted to this subject. They ordinarily produce no symptoms, but it is to be noted that they appear to be more frequent in cases of diarrheal disturbance, and possibly have some etiological connection with this condition.

CHAPTER IX

DISEASES OF THE LIVER, GALL-BLADDER AND PANCREAS

Anatomical and physiological characteristics of the liver as compared with those obtaining in adult life have already been referred to under the Normal Anatomy and Physiology of the Child (p. 41). Diseased states of the liver, both functional and organic, are on the whole decidedly less frequent in early life than later.

FUNCTIONAL DISTURBANCE OF THE LIVER

(Biliousness; Bilious Indigestion)

Of this condition, to which the old designations as given above have long been applied, nothing positive is known. Yet it seems beyond question that, combined with functional disorders of other portions of the alimentary apparatus, the liver is also involved. In some cases the stools are constipated, or perhaps diarrheal, in character, and of too light a yellow color, or even whitish showing the absence of a normal amount of bile. In how far the various dyspeptic sensations present, such as loss of appetite, nausea, vomiting, malaise, flatulence, a bitter taste in the mouth, sallow tint of the skin, headache, and the like, depend upon the liver, and to what extent upon other organs, especially the intestine, it is impossible to determine. The treatment is that of intestinal indigestion.

CONGESTION OF THE LIVER

This may be *active* or *passive*. The former sometimes occurs in acute fevers, from overeating, and from the ingestion of too rich a diet. Passive congestion is more frequent, and is seen in chronic diseases of the heart and lungs, chronic pleurisy, chronic malaria, or other causes which interfere with the normal venous circulation in the liver or in the system in general.

¹ Wien. klin. Rundschau, 1913, XXVII, 387.

² Ziegler's Beiträge f. path. Anat. und allg. Path., 1914, LVII, 474.

The **symptoms** vary with the cause, and are not characteristic. The liver increases in size, both in an upward and a downward direction, and its hard edge may be felt decidedly below the normal position. In the acute condition there may be pain and tenderness in the hepatic region and perhaps slight jaundice; in the chronic cases there may also be moderate jaundice, evidences of indigestion, and the symptoms characteristic of the cause. When attendant upon cardiac disease, a rapid diminution in the enlargement may follow improvement in the state of the circulation. This diminution is of the portion of the swelling which is due to an acute engorgement. The remaining portion, dependent upon alterations of the hepatic tissue, naturally remains unchanged. The treatment is that of the cause.

ICTERUS

(Jaundice)

This is a symptom due to many causes. That designated *icterus neonatorum* and that due to *congenital obliteration of the bile-ducts* have already been discussed under Diseases of the New Born (pp. 273, 274), where also certain other conditions occurring at that period and accompanied by icterus have been referred to. There remain to be considered a number of varieties of icterus seen in infancy or in childhood.

The classification of the forms of jaundice, itself but a symptom, is to an extent unsatisfactory since it is still not thoroughly understood how the symptom is produced in every case. Some investigators would group most of them together as instances of *infectious* icterus. Others denominate the majority *obstructive*, on the ground that the discoloration is caused by interference with the passage of bile, the result of a catarrhal swelling of the larger or smaller ducts, whether or not this swelling has to do with an infectious process. There is a tendency also to widen the class of *hemolytic* cases, in which a hemolysis within the vessels is the primary feature. It is certain only that obstruction is the primary cause in some instances, infection in others, and hemolysis the prominent feature in the third group; but many cases cannot be classified with accuracy, and may, too, belong to more than one category. The matter is so far from final settlement that any classification can be but tentative.

(1) CATARRHAL ICTERUS OF INTESTINAL ORIGIN

(Obstructive Jaundice)

This form of icterus is often discussed in the class of gastroenteric affections under the title of *duodenitis* or *gastroduodenitis*. Its prominent symptom, however, makes it conveniently considered with hepatic diseases.

Etiology.—The jaundice follows a gastroduodenal catarrh, and is attributed to an extension of the swelling of the mucous membrane of the duodenum to the opening of the common bile-duct or into it, closing it by a catarrhal inflammation. To this theory it has been with reason objected that, were the jaundice dependent upon catarrhal swelling, it should be much more frequent in early life than it actually is; and it is possible that the cause may be of an infectious nature. In fact a tendency to the epidemic occurrence of this mild form of icterus is sometimes seen, and allies this variety very closely with that next to be described. The disease is comparatively infrequent in children and uncommon in

infancy. I have seen it in the latter period in a relatively few instances.

Symptoms.—The only characteristic one is the yellow coloration of the skin and mucous membranes and of the urine. Before the actual symptoms of jaundice appear some degree of indigestion usually exists for a few days. This may be so mild that it is overlooked, or severe enough to produce repeated vomiting, fever, abdominal pain, and constipation or sometimes diarrhea. A few days later icterus develops. This is usually slight at the beginning and is first seen in the conjunctivæ, but increases until in a day or two it is well-developed over all the visible mucous and cutaneous surfaces. It is seldom, however, of the deep bronze-yellow tint observed in some jaundiced conditions in adults. The appetite is diminished or normal, the tongue coated, the breath heavy. The urine is often dark-colored from bile, even before the yellow tint of the skin is apparent. The stools are more or less acholic, having a putty color, the degree of change depending upon the completeness of the obstruction. Headache, fretfulness, and malaise are frequent. The abdomen is often distended with gas, and there may be tenderness in the hepatic region. Itching of the skin and slowness of the pulse may occur in the severer cases, but are not common. The liver is generally slightly enlarged and the spleen sometimes so; but an enlargement of this latter suggests that the case is not one of simple catarrhal jaundice.

Course and Prognosis.—The prognosis is entirely favorable, but the course is sometimes somewhat prolonged, the icterus slowly fading but not disappearing completely for 2 or 3 weeks. The acute digestive symptoms generally cease in a few days after the onset.

Diagnosis.—This is seldom difficult. The association of the condition with dyspeptic disturbances is an indication of the cause, and the benign course and comparatively short duration remove doubt. In occasional cases in early infancy the disorder is to be distinguished especially from icterus neonatorum, septic icterus, and from such forms as congenital family icterus. It is differentiated especially by the greater degree of jaundice and the presence of bile in large amount in the urine. In rare cases in early infancy obstructive jaundice may occur from malformation of the bile ducts. The course of the case will settle this question in a short time.

Treatment.—This consists principally in correcting the diet. Since during jaundice the alimentary tract finds much difficulty in the digestion of fats, these should be reduced as far as possible. The starches too are often a cause of intestinal indigestion, and this element of the food should be given in somewhat diminished amount. Feeding with skimmed milk and broth free from fat is indicated; later with meat, when the acute febrile symptoms have disappeared. Water should be given freely, especially some alkaline water such as Vichy. Purgatives every day or every other day are of value, calomel being popular for this purpose, but effective not because it exerts any specific action upon the liver. In other cases the daily administration of a saline laxative is of service, such as sulphate of magnesia alone or combined with rhubarb. The employment daily or every other day of large enemata of tepid water is an aid in many cases. Such symptoms as abdominal pain, vomiting and the like require treatment especially directed to these. (See Gastritis, p. 721.) The administration of mineral acids has long been popular, but I have never been able to satisfy myself that they are of any special value.

(2) ICTERUS OF INFECTIOUS OR HEMOLYTIC ORIGIN (see also Vol. II, p. 489)

Simple catarrhal jaundice shades almost imperceptibly into the type now to be considered. This is of wide variety and may be made to include, for the sake of convenience, both those cases which are clearly infectious in origin, and those where it is known that hemolysis within the circulation has taken place; the latter condition being sometimes consecutive to the former; sometimes occurring independently of it. The probability is that all, or nearly all, instances of this form of icterus are in the final event and in the broader sense really obstructive. This may be the result in some cases of choking of the minute intrahepatic blood-vessels with released blood-coloring matter, or of the capillary bile-ducts with thick bile; both resulting from the hemolysis in process; or in other cases perhaps being caused by swelling of the biliary passages (angiocholitis) the product of the infectious process. They are not usually, however, obstructive in the narrower sense. There is no sharp differentiation possible between some of the cases believed with reason to be infectious and others of the obstructive catarrhal class; nor can we make a sharp distinction between infectious and hemolytic icterus, inasmuch as the hemolysis very probably in many cases has an infectious origin. There is also a very close relationship between some of the cases of hemolytic icterus and forms of splenomegaly (Krumphaar).¹

Etiology.—The causes are various. The cases may develop in the course of any of the acute infectious diseases, such as malaria, typhoid fever, scarlet fever, pneumonia, and sepsis. The disease has also repeatedly been known to occur in local and sometimes widespread epidemics; a strong indication of its infectious nature. A large number of such outbreaks in England are quoted by Guthrie.² The very severe forms of icterus known as *icterus gravis* and as *Weil's disease* are also clearly of an infectious nature in many instances. Pernicious anemia is likewise a cause, the icterus being the result of the hemolysis which is taking place. The nature of the germ producing the infection is not clearly understood. Doubtless more than one species have the power to cause it.

Symptoms.—There is great variation in the intensity of the symptoms in the different forms of infectious icterus which are included under this heading. In many cases the discoloration of the skin is less marked than in the catarrhal icterus previously described, and the stools still contain bile and the urine none (*acholuric icterus*). This is true especially of icterus resulting from septic poisoning. (See Sepsis of the New born, and Pernicious Anemia, p. 258, and Vol. II, p. 462.) In other instances the jaundice may be intense and the urine exhibit bile. The symptoms in general are little characteristic, and often overshadowed by those of the primary diseases. The amount of bile in the urine varies with the case and with the cause. A slight albuminuria is not infrequent. In the severest cases of infectious icterus (Weil's disease) there are intense discoloration of the skin, enlargement of the liver and spleen, high fever, hemorrhage from the mucous membranes or into the skin, nephritis, delirium, and convulsions or coma. In the worst cases death may take place in the course of a very few days. Instances of this sort are rare in children. In some cases of hemolytic icterus (Hayem Type)³ there is a

¹ Journ. Experim. Med., 1912-14, various references; Amer. Journ. Med. Sci., 1915, CL, 227.

² Brit. Jour. Child. Dis., 1913, X, 1.

³ Presse méd., 1898, V, 121

chronic icterus with bile in the blood-serum but not in the urine, and no symptoms of obstructive jaundice are present. There is anemia and enlargement of the liver and spleen. In none of Hayem's cases was there any family history of icterus. The *prognosis*, *diagnosis* and *treatment* of forms of infectious and hemolytic icterus are those of the cause. (See also Vol. II, p. 489.)

(3) CONGENITAL AND FAMILIAL ICTERUS

(Congenital Acholuric Jaundice; Congenital Hemolytic Icterus;
Congenital Family Cholemia)

Here might be grouped a class of cases characterized by the tendency to family incidence often seen, and the very early period at which the symptoms appear in some of them. Occasionally infants are born already jaundiced, or become so very promptly; have a very distinct familial history of the disease; are evidently much more ill than in the ordinary cases of icterus neonatorum, which has already been discussed, and exhibit a discoloration very persistent, although finally disappearing. I have seen a number of such cases, some with a distinctly hemorrhagic tendency. In speaking of congenital icterus, no reference is made here to the rare and severe cases of jaundice depending upon obliteration of the bile-ducts or congenital hepatic cirrhosis, or to the cases in the new born clearly the result of sepsis.

As to the family incidence, some remarkable examples have been reported, as for instance those of Pearson¹ in which 10 of 11 children born died of jaundice soon after birth; and of Arkwright² where 14 out of 15 children born of one mother suffered jaundice soon after birth, and 10 died. The mother had had icterus at the age of 4 years. A number of other series have been published, among them a family reported by Hutchinson and Panton³ in which the disease had occurred in 3 generations.

It is very probable that cases of familial icterus may be unlike in nature and origin. Minkowsky⁴ reported 8 cases occurring in the new born through 3 generations, and drew attention to the fact that the icterus was of the acholuric variety, the urine exhibiting urobilin but no biliary coloring matter. This type frequently bears his name, and the following description applies in large part to it.

Etiology.—The nature of the cause is not at all understood, but it seems probable that there is a defect in the blood-forming functions of the body, and also that an increased destruction of the corpuscles takes place in the spleen. The influence of heredity has already been spoken of. Other diseases seem to play no part. In older children chilling or over-fatigue has seemed in some cases to be the immediate cause.

Symptoms.—The symptoms may be present at birth or appear soon after it; or in another class of familial cases develop only after the patient reaches childhood or even adult life. The degree of jaundice varies with the case, and even in the same individual from time to time. It is usually not great, and sometimes is very slight and is of an acholuric character, the urine, although dark-colored from urobilin, being free of biliary coloring matter, or showing only at times a small amount, and the feces containing bile. As pointed out by Chauffard⁵ there is increased

¹ Underwood's Diseases of Children, 1846, 168.

² Edin. Med. Jour., 1902, LIV, 156.

³ Quart. Jour. of Med., 1909, II, 432.

⁴ Verhandl. der Kong. f. inn. Med., 1900, XVIII, 316.

⁵ La sem. méd., 1907, XXVII, 25.

fragility of the corpuscles to salt solutions in this congenital hemolytic jaundice, one of the normal strength producing separation of the blood-coloring matter. This fragility is in sharp contrast to the condition found in ordinary obstructive jaundice, in which there is an increased resistance of the red blood-corpuscles to salt solution. Bile-pigment is generally present in the blood-serum. The spleen is always enlarged. There is anemia of moderate degree, with a few megaloblasts and normoblasts and many reticulated red cells, reaching even as high as 20 per cent. of the erythrocytes. The number of leucocytes is not altered. There is sometimes seen a tendency to hemorrhage, but not to the extent characteristic of the primary hemorrhagic diseases. As the patient grows older the general health may be but little affected, except perhaps during the exacerbations. There are no digestive disturbances, and no abdominal tenderness or pain.

Course and Prognosis.—The prognosis is uncertain. In early infancy many die soon; but should this not happen the later course seems to be without influence upon the general health. The jaundice, however, never entirely disappears.

Diagnosis.—This rests especially upon the family history, when obtainable; the moderate degree of jaundice; the fragility of the corpuscles; the beginning in infancy usually seen; the absence of bile from the urine and its presence in the feces; and the enlargement of the spleen. *Biliary cirrhosis* of the liver is of later development, generally without familial history, and there is a greater degree of jaundice. *Banti's disease* (Vol. II, p. 486) is primarily a disorder of the spleen, the jaundice being generally a later development, and no increased fragility of the corpuscles being present.

Treatment.—For most of the cases this is symptomatic merely. Splenectomy has been done with success, but should not be employed unless the general condition of the patient is suffering.

(4) ICTERUS FROM OTHER CAUSES

Jaundice appears in many other conditions than those mentioned. In some it is but a mild symptom; in some, although a prominent one, it is grouped with other features in such a way that a separate description of the complex is necessary. Typical obstructive jaundice, not of a catarrhal nature, occurs in childhood rarely from the presence of impacted gall-stones, the pressure of a malignant growth upon the bile ducts, or the wandering of a worm into these. The ingestion of such poisons as arsenic and phosphorus or the accident of a snake bite are possible causes of icterus of a hemolytic nature. Cirrhosis of the liver may have icterus as a symptom, or it may occur in the course of hepatic abscess, or of some form of splenic enlargement or of anemia, and is very well marked in acute yellow atrophy, and in the acute infectious hemoglobinemia already described (p. 262) among diseases of the new born.

(5) ACUTE YELLOW ATROPHY OF THE LIVER

This disease, one of the forms of icterus gravis, uncommon at any time of life, is very rare in childhood and infancy. Phillips¹ was able to collect 41 cases in children, including 4 of his own; and Francioni² places the number at 46, and to these perhaps half a dozen more might be added. One of the youngest of the series was in an infant of 4 days (Politzer).³

¹ Amer. Journ. Med. Sci., 1912, CXLIH, 177.

² Riv. di clin. pediat., 1914, XI, 653.

³ Jahrb. f. Kinderh., 1860, III, 10.

Twice as many boys as girls are attacked; in contradistinction to adult life, in which more women suffer, I have seen 2 cases in later childhood, one of which, in a boy of 7 years, I have previously reported.¹

The usual *pathological lesions* are found at autopsy; the liver being shrunken perhaps to half its size, with a more or less wide-spread parenchymatous degeneration present. Efforts at a reparative process in the liver have been discovered. The causes and symptoms are exactly as in adult life. The former are entirely unknown, although the occurrence of acute digestive disturbances, acute febrile diseases, and syphilis have sometimes been in existence, and may have predisposed in some way. The disease is ushered in by icterus of an ordinary type, which may last a few days or 1 or 2 weeks. After this there develops rapidly high fever, hemorrhages, vomiting, and such nervous symptoms as convulsions, delirium, or coma as in other forms of severe icterus. In addition there is a progressive diminution in the size of the liver with local tenderness, and death takes place in 3 or 4 days. The *diagnosis* rests upon the grave general symptoms, the presence of intense jaundice, the diminution in the size of the liver, and the occurrence of bile and of leucin and tyrosin in the urine. It is difficult to understand how patients with the serious alterations of the liver, which this disease shows at autopsy, could survive, and an element of doubt must attach to the few reported instances of recovery, although some appearing to be reasonably certain instances are on record. Fletcher² reports a case recovering after repeated hypodermoclysis.

There are certain cases described as *subacute atrophy*. Chisholm³ details 9 cases which he had collected from medical literature, Wegerle⁴ 2 cases, and Fraser⁵ 1 case. The causes are probably different from that of the acute disease, varying with the case, and sometimes are distinctly discoverable at autopsy; cirrhosis, tuberculosis, and infection being among them. The symptom always present was jaundice; but in other respects the clinical manifestations were not characteristic. Leucin and tyrosin were not found in the urine in any. The disease lasted several weeks. All of the cases reported have terminated fatally.

ENLARGEMENT OF THE LIVER

Increase of the size of the liver is very frequent in early life, being a symptom in a large number of diseased conditions. Among these may be mentioned rachitis; congenital syphilis; tuberculosis; congestion, especially from heart disease; fatty and amyloid degeneration; tumors; hydatid cysts; cirrhosis; and certain diseases of the blood and the blood-making organs. The diagnosis of these various conditions is made by consideration of the symptoms exhibited by other organs. The disorders now to be described have hepatic enlargement as a symptom at some period of their course.

CIRRHOSIS OF THE LIVER

This is an uncommon disease in early life. I recall seeing but 2 cases, one an instance of typical alcoholic cirrhosis in a girl of 11 years; the other a probable biliary cirrhosis in a child of 6 years. Collective studies

¹ Arch. of Ped., 1899, XVI, 330.

² Garrod, Batten and Thursfield, Diseases of Children, 1913, 210.

³ Brit. Jour. Child. Dis., 1914, XI, 397.

⁴ Frankfurter Zeit. f. Pathol., 1914, XV, 89.

⁵ Amer. Journ. Med. Sci., 1916, CLII, 202.

of the affection as observed in children have been made by Howard,¹ Edwards,² Jones³ and others.

Etiology.—The disease may attack children of any age, but appears commonest in later childhood. Jones records cirrhosis 33 times in 17,891 autopsies on children, giving an incidence of 0.185 per cent. In Edwards' series of 100 cases 65 were from 6 to 16 years old. The majority of the reported instances have been in males (Edwards, 53 males; 33 females; 14 not stated). The causes are various, but are probably oftenest of a toxic or infectious nature. The disease appears in some instances to follow the acute infectious fevers, but the real influence of these is doubtful. According to most statistics the misuse of alcohol has been the cause in a not large percentage in early life. Thus in Edwards' series of 100 cases there was a positive history of the ingestion of alcohol in only 11. In Jones' collection of 300 cases, however, representing different types of cirrhosis, but not including the cases of Ghose,⁴ there were 74 instances of alcoholic cirrhosis. Syphilis appears to be the causative factor in few instances; and although it is probable that the majority of those seen in early infancy depend upon a syphilitic process, it is very likely that in most of those designated as cirrhosis the pathological lesions are not purely of a cirrhotic nature. Malaria and tuberculosis are also probable causes, and chronic passive congestion from disease of the heart, polyserositis, or other agents gives rise to the disorder. The occurrence of a congenital biliary cirrhosis has already been referred to in connection with congenital obliteration of the bile ducts (p. 273). This is the form of cirrhosis oftenest seen in the new born, but is rare. Biliary cirrhosis is met with more frequently in later childhood than before this period. A familial tendency has also been observed in cirrhosis of both the portal and biliary types. An interesting family history with 4 children affected is reported by Bramwell.⁵

Not included in the statistics quoted appears to be a special form of biliary cirrhosis reported as not infrequent in infants in hot countries, which exhibits a decided familial tendency. This is stated by Ghose to be very common in India, where he has observed 400 cases, of which only 6 recovered.

Pathological Anatomy.—This is entirely of the nature seen in adult life, with the exception that shrinking of the size of the organ at this period is less frequent. This is probably because the cause is less often the misuse of alcohol, and the process is, therefore, not so often in children an atrophic or portal cirrhosis, as that of the hypertrophic or biliary form. In the cases of *portal* origin there is, namely, generally a diminution in the size of the liver, its surface is uneven, and section shows an irregular distribution of connective-tissue-hypertrophy compressing and penetrating the lobules, and producing degeneration and atrophy of the cells. In the *biliary* type, or hypertrophic cirrhosis (Hanot's cirrhosis), the spleen and liver are enlarged, the latter being greenish or yellowish in color, and on section showing an interlobular overgrowth of connective tissue compressing the small bile-ducts, and an inflammation of these. In another type of hypertrophic cirrhosis, depending upon the chronic passive congestion of cardiac disease, especially

¹ Amer. Jour. Med. Sci., 1887, XCIV, 350.

² Arch. of Ped., 1890, VII, 502.

³ Brit. Jour. Child. Dis., 1907, IV, 1.

⁴ Lancet, 1895, I, 321.

⁵ Edin. Med. Journ., 1916, XVII, 90.

chronic pericarditis, the *nut-meg liver* is produced. To the evidences of engorgement in this form is added a moderate development of fibrosis under the capsule and unevenly throughout the liver. The spleen is enlarged.

Although this description represents the types of pathological changes seen, mixed forms are common, the cirrhotic changes occurring often irregularly. The pathological alterations observed in the liver in hereditary syphilis may be clearly cirrhotic, showing an interstitial hepatitis, and this may be further combined with scattered gummata of various size. (See Syphilis of the Liver, pp. 565, 573, 577.)

Symptoms.—These do not differ greatly from those seen in adult life. The early symptoms are modified, but point to a digestive disturbance, with loss of appetite and not infrequently diarrhea and occasional vomiting, followed later by progressive emaciation with a sallow pallor, more or less dilatation of the abdominal veins, enlargement of the liver and spleen, and ascites. Albumin may appear in the urine if a complicating nephritis develops. Later, the liver may grow smaller than normal (*atrophic form*), or it may remain enlarged (*hypertrophic form*). Hemorrhages from the skin or mucous membranes are uncommon, and the temperature is irregular; there being sometimes attacks of fever with abdominal pain and sometimes a subnormal temperature. In the atrophic form of the disease jaundice is absent or slight and ascites is common; while in biliary cirrhosis the icterus is often intense, the urine contains bile, the stools are not acholic, the spleen is greatly enlarged, and ascites is less frequent. The symptoms of congenital biliary cirrhosis have already been described (p. 273).

Course and Prognosis.—The course is often more rapid than in adults, although the disease sometimes lasts several years. The prognosis is unfavorable, death taking place from exhaustion, often with diarrhea and sometimes with delirium, coma, or convulsions. Pulmonary complications may develop and be the direct cause of the fatal ending. In cases of cirrhosis of any type of long duration the growth of the body may be retarded. In the hypertrophic form exacerbations of symptoms occur from time to time, with renewed fever and pain, followed by periods of quiescence.

Diagnosis.—Early in the disease there are no certain data upon which a diagnosis can be based; later, enlargement of the liver and spleen, jaundice, and attacks of fever and abdominal pain, make the diagnosis of biliary cirrhosis probable. *Banti's disease* (see Vol. II, p. 486) is distinguished by the leucopenia, the lesser degree of jaundice and the predominating and early splenic enlargement. *Congenital hemolytic icterus* is recognized by the absence of bile from the urine, the much less degree or absence of enlargement of the liver, and the increased fragility of the red blood corpuscles. The presence of ascites is oftener dependent upon a *tuberculous peritonitis*; and it is only if the liquid is removed by tapping that an abnormal diminution in the size of the liver may be determined and the diagnosis of an atrophic cirrhosis made, or a hypertrophic cirrhosis found associated with chronic pericarditis or mediastinitis. The occurrence of hemorrhages is a suggestive symptom of cirrhosis.

Treatment.—This can be but palliative, with the intent of prolonging life. The diet should be as unirritating as possible, milk being most useful for this purpose. Should there be any suspicion of the existence of syphilis, treatment for this disease should be given. This is

the only form of cirrhosis in which permanent benefit by drugs may be hoped for. On the ground that the symptoms may be due to congestion and not to cirrhosis, purgatives should be tried; and if disease of the heart is present, remedies should be given appropriate to this. As in the case of adults, aspiration of the fluid is sometimes necessary. Surgical measures to establish collateral circulation have repeatedly succeeded in adults, and recovery followed in the case of a boy of 6 years with alcoholic cirrhosis reported by Grosz.¹

FATTY LIVER

Fatty infiltration of the liver is a common affection in early life, especially in infancy. Freeman² found it in 40 per cent. of 496 autopsies in children; and his studies show, too, that there was no special relationship of this lesion to any one other disorder, although it was oftener present in acute infections than in chronic wasting diseases. The liver is large, smooth, and in well-marked cases of a decidedly yellow color, and is distinctly greasy on section. Wollstein³ found fatty liver in a surprisingly large number of cases dying of tuberculous diseases (45 in 67 cases). In fatty *infiltration*, which is the common form, the hepatic cells are infiltrated with fat, but the cell-nucleus is unaltered. When fatty *degeneration* is present, the result of toxic or infectious processes, the cells and nuclei undergo a degenerative process, and become granular and cease to stain normally. Apart from the enlargement there are no special symptoms of fatty infiltration which make it possible to recognize it with certainty during life.

The prognosis and treatment are that of the disorder producing it, as far as this can be ascertained.

AMYLOID LIVER

Amyloid degeneration of the liver is the result of a number of conditions, and is associated with similar degeneration in other organs, such as the kidneys, spleen and intestine. Chronic suppurative processes are prominent among the causes. Consequently, amyloid liver is seen in tuberculous disease of the bones and lymphatic glands, and in long-continued empyema and bronchiectasis. It may occur also as a result of syphilis and rickets. The increase in the size of the liver is often greater than in any other form of hepatic enlargement. The organ is smooth, waxy and glistening in appearance, hard, firm, and gives the ordinary amyloid-red reaction with iodine. The walls of the arterioles first suffer from the degeneration and then the hepatic cells.

The symptoms are indefinite and are largely those of the causative primary disorder. There is a very striking waxy pallor of the face, emaciation is common, diarrhea is frequent, and the digestion in general much impaired. Albuminuria and general dropsy are not uncommon, depending upon the complicating amyloid involvement of the kidneys or upon pressure of the very large liver upon the abdominal vessels. Unless produced by such causes, ascites is infrequent. There is generally no icterus, abdominal pain or tenderness. The much enlarged, smooth liver and spleen can be readily felt through the abdominal walls.

The course of the disease is slow, and the prognosis is unfavorable, although not invariably so if the causative disease can be arrested. Death occurs from exhaustion or from some intercurrent affection.

¹ Ref. Monatssehr. f. Kinderheilk., 1903, II, 386.

² Arch. of Ped., 1900, XVII, 81.

³ Amer. Jour. Med. Sci., 1902, CXXIII, 817.

The **diagnosis** is based upon the occurrence of very large liver and spleen, the peculiarly waxy pallor, and the presence of some primary affection capable of producing the degeneration. Treatment is purely that of the cause.

ABSCESS OF THE LIVER

(Suppurative Hepatitis)

This is a very uncommon condition in early life. The disease at this period has been studied especially by Musser¹ in 1890, who could collect but 34 cases; and more recently by Legrand² who has increased the number to 122. Even in tropical countries the latter writer found abscess uncommon in early life.

Etiology.—The causes are the same as in adults, among them being trauma; suppurative processes of the bones, appendix, peritoneum, etc.; the infectious diseases; dysentery; and pulmonary tuberculosis. In addition, in infancy, abscess is due to the wandering of ascarides into the bile-ducts, and exceptionally is dependent upon sepsis connected with the umbilical vessels. In Legrand's series 31 per cent. of the cases depended upon dysentery; 19 per cent. on trauma; 15 per cent. on appendicitis; 6 per cent. on typhoid fever; 10 per cent. on tuberculosis; 13 per cent. on intestinal worms and 9 per cent. on pyemia. The abscess may be single, as when resulting from trauma; or oftener multiple, as when of septic origin, the immediate cause then being a suppurative pylephlebitis. Occasionally pus from a hepatic abscess collects between the diaphragm and the liver (*subphrenic abscess*). It is to be noted, however, that abscess in this region may arise in other ways.

Symptoms.—The symptoms of solitary abscess consist of pain and tenderness in the hepatic region; enlargement of the liver either upward or downward; fever of a hectic type with chills and sweats; emaciation; loss of strength; vomiting; diarrhea; loss of appetite; leucocytosis, and sometimes slight jaundice. In some instances the pain is not in the region of the liver but in the chest, the right shoulder, or the abdomen. There may be painful respiration, and cough or shortness of breath if the disease is in the upper portion of the liver. When the symptoms described are associated with a history of an injury in the hepatic region, occurring perhaps some time before, the presence of abscess is very probable. Only a successful aspiration can make the diagnosis certain. The symptoms of multiple abscess are less definite. If, following the existence of a known suppurating focus, there develop painful enlargement of the liver, jaundice, fever of a hectic type, diarrhea, and typhoid symptoms, the existence of multiple abscesses may be suspected.

Course and Prognosis.—These vary with the nature of the affection. The duration is generally from 1 to 2 months. If in the case of single abscess the pus can be reached by operation, the chance of recovery is fair, depending, however, upon the seat. When in the upper portion close to the diaphragm the prognosis is not favorable. Multiple abscesses always give a most unfavorable prognosis. **Treatment** is entirely surgical, aspiration or incision being required.

MORBID GROWTHS OF THE LIVER

Tumors of the liver are not of common occurrence in early life. They have been studied especially by Steffen³ who collected 39 cases of primary

¹ Cyclop. Dis. of Child., Keating; 1890, III, 466.

² Arch. de méd. des enf., 1906, IX, 129.

³ Malign. Geschwülste im Kindersalter, 1905, 77.

malignant growths. Carcinoma, adenocarcinoma, and sarcoma may be mentioned among neoplasms of this class. In the majority of cases the tumor is secondary to one elsewhere in the body, but in a considerable number it is primary. It is noteworthy that in early life carcinoma appears to be more frequent in the liver than in other localities. I¹ have observed 1 case of primary carcinoma in an infant of 21 months, and have been able to collect in all, including this, 55 instances of primary carcinoma of the liver in early life. Among nonmalignant growths are congenital multiple cysts, lipoma, fibroma, adenoma and angioma. Either of the last two may be multiple or single. Veeder and Austin² describe a case of multiple congenital hemangio-endothelioma, and refer to 3 others recorded in medical literature.

The **symptoms** of growths of the liver consist in steadily increasing size of the organ, which often exhibits irregularly nodular masses increasing debility; abdominal pain; and the various symptoms depending upon pressure. Yet ascites and icterus may be absent until, perhaps, the latest stages.

Among new growths may conveniently be placed **tuberculosis of the liver**, always secondary to tuberculous lesions elsewhere. Only occasionally large, cheesy masses are found, but miliary tubercles on the surface of the liver or scattered through the organ are of common occurrence.

ECHINOCOCCUS OF THE LIVER

(Hydatids)

This is of very exceptional occurrence in infants and children, and this is especially true of North America. The causes and symptoms are the same as in adult life. The parasite is the echinococcus-form of the *tænia echinococcus*, a minute tapeworm occurring in the dog. If by the ingestion of contaminated food or water the eggs by chance enter the stomach of man or other animals, they produce embryos which penetrate the blood-vessels and lymph-channels and lodge oftenest in the liver. Here the parasite develops into a cyst, which increases in size independently of the production of daughter-cysts. The principal symptom is the enlargement of the hepatic area which the cysts produce. This may be discovered by percussion and palpation, the latter sometimes giving a sensation of fluctuation and a "hydatid fremitus." Aspiration reveals a clear fluid in which the hooklets of the parasite are found. Other symptoms of various sorts may develop, depending upon pressure in different directions; and in this case are similar to the pressure-symptoms which may be produced by hepatic growths of any nature. Occasionally rupture of the cyst may occur, or sometimes suppuration take place in it and produce evidences of sepsis. The prognosis is always doubtful. Spontaneous recovery may take place through simple shrinking of the cyst, or through external rupture and discharge; but this is rare. Oftener death occurs unless treatment is instituted. This latter is entirely surgical in nature.

DISEASES OF THE GALL-BLADDER AND BILE-DUCTS

Conditions of this nature are very rare in early life. Congenital obliteration of the bile-ducts has already been described. (See p. 273.) Only exceptionally there exists a congenital absence of the gall-bladder. Sev-

¹ Amer. Jour. Med. Sci., 1918, CLX, 79.

² Amer. Jour. Med. Sci., 1912, CLIII, 102.

eral such reported cases have been collected by Eshner.¹ Occasionally a round worm penetrates into the bile-duct and sets up an inflammation there. Acute cholangitis and cholecystitis are very infrequent conditions, but may occur after typhoid fever or from sepsis. Cholecystitis of a more chronic nature is probably not an infrequent sequel of typhoid fever. Tuberculosis of the gall-bladder is sometimes seen, and that of the bile-ducts is not uncommon, producing small nodules or cavities within the liver. Very exceptionally gall-stones are discovered. Thomson² collected 6 reports of gall-stones in the new born and added another. Still³ found in medical literature 20 cases of gall-stones in early life, to which he added 3 of his own. In all of these the stones were found in the feces, or in the gall-bladder at autopsy; 10 were in infants still-born or dying in a few weeks; 4 in those from 3 to 9 months of age; 1 in an "infant"; 8 were in children from 3 to 14 years old. Khautz⁴ could collect but 15 cases from medical literature, 6 of these being in infants.

DISEASES OF THE PANCREAS

Organic disease of this organ plays a very minor rôle in early life. Syphilitic involvement with the production of gummata and increase of the connective tissue and atrophy of the glandular substance is occasionally seen. Tuberculous nodules of considerable size, or an infiltration by many small nodules, may accompany a general tuberculosis of the body of the patient, and is not at all infrequent. Amyloid degeneration may occur in conjunction with this change in other organs. Tumors also may involve the pancreas, being either primary, or secondary to morbid growths elsewhere. Among the former primary sarcoma has been described by Litten⁵ and others. Cysts are occasionally found, oftenest of the class of retention cysts. Calculi and ascarides have been reported present in the pancreatic duct. An **acute pancreatitis** is occasionally seen as one of the manifestations of mumps. (See p. 500.) Even this is infrequent; and dependent upon other causes pancreatitis is of great rarity, but may occur in the course of infectious diseases; as a result of the presence of round worms in the duct; by infection extending from the intestine; or be metastatic in origin. The inflammation may be either hemorrhagic or suppurative. Although abdominal pain and tenderness rapidly followed by collapse occur as symptoms, these are not sufficiently distinctive to allow of an diagnosis being made. A **chronic pancreatitis** is distinctly more common, producing a fibrosis of the organ. The most frequent cause is hereditary syphilis, although the disease may also accompany gastroenteritis. It is seen also in diabetes mellitus. It is possible that some of the cases of fatty stools depend upon this form of pancreatitis.

How often a functional disturbance of the pancreas may account for difficulty in digestion is not known, but it is very probable that some of the instances of fat-indigestion may be due to this. Some cases of infantilism are associated with disturbance of the functions of the pancreas. (See Infantilism, Vol. II, p. 533.)

¹ Med. News, 1894, LXIV, 548.

² Edinb. Hosp. Rep., 1898, V, 1.

³ Transac. Path. Soc. London, 1899, I, 151.

⁴ Centralbl. f. d. Grenzgeb. der Med. u. Chir., 1913, XVI, 545.

⁵ Deut. med. Wochenschr., 1888, XIV, 901.

CHAPTER X

DISEASES OF THE PERITONEUM

ACUTE PERITONITIS

In the great majority of cases, peritonitis is secondary to a lesion elsewhere in the body. An acute primary peritonitis does, however, sometimes occur, although much less frequently than the secondary form.

Etiology.—Age exerts a decided influence. The disease may occur before birth as a result of hereditary syphilis. In the new born peritonitis is not uncommon, depending upon septic infection usually from the umbilicus. In infants after this period it is rare, and becomes frequent again only with the increasing incidence of appendicitis. This latter condition is much the most common cause of a secondary peritonitis in childhood, the inflammation either following a perforation or resulting from extension of the process from the perityphlitic inflammation.

Very rarely in early life perforation of a gastric or duodenal ulcer, or the rupture of an abscess in some other abdominal organ, gives rise to secondary peritonitis. I have seen it, for instance, consecutive to perforation of a duodenal ulcer in a child of 5 months; in another of 19 months to a diverticulitis; and in a considerable number of instances under my observation it has followed intestinal perforation in typhoid fever. In other cases peritonitis may be produced by the rupture of an empyema, or of an abscess resulting from spinal caries or from inflammation about the kidney. Strangulation of the intestine, as in hernia and intussusception, may likewise cause the disease, as may also forms of enteritis. Inflammation of the female genital tract is an occasional cause, although with so much less frequency than in adult life that it is entirely exceptional. Trauma, such as blows received on, or operation done upon, the abdomen, and exceptionally exposure to cold, are sometimes followed by a primary peritonitis. Acute infectious diseases may produce a peritonitis dependent upon the infection. Here especially are to be mentioned erysipelas, grippe, scarlet fever, typhoid fever, and diphtheria; while pleurisy and pneumonia may have peritonitis in their train.

Every case of acute peritonitis is directly dependent upon the action of some species of microorganism, either alone or often in combination with other species. The streptococcus is the most frequent cause in cases depending upon sepsis in the new born, or in those following scarlet fever, tonsillitis, erysipelas and some other infectious diseases. In some instances a streptococic peritonitis appears to be primary. The pneumococcus is capable of producing a primary peritonitis or may be the agent in cases consecutive to pneumonia or pleurisy. This pneumococic peritonitis is more frequent in early than in adult life. Barling¹ collected 234 reported cases occurring in children. Of these 62 were males and 172 females. The infection is probably oftenest through the blood-channels, although Annand and Bowen² would assign the majority of cases to infection from the intestinal tract. In perhaps the greater number of instances of peritonitis the bacillus coli is found present, very frequently in combination with some other species. Other cases exhibit the staphylococcus aureus, the bacillus pyocyaneus, the proteus vulgaris, or other germs. The gonococcus is the cause of peritonitis oftenest in cases following vulvovaginitis.

¹ Practitioner, 1912, LXXXVIII, 537.

² Lancet, 1906, I, 1591.

Pathological Anatomy.—The lesions are not different from those seen in adult life. There is at first redness and dullness of the peritoneum followed by exudation of fibrinous and purulent serum. In some instances the fibrinous element predominates; in others the serous or the purulent. In the first, patches of yellowish lymph are found upon the abdominal wall and upon the peritoneum covering the intestines, uniting these latter together more or less firmly according to the duration of the case. In those recovering firm adhesions may remain. In cases where the serous element predominates there is a considerable effusion of yellowish, clear or slightly cloudy, serous fluid in addition to the plastic lymph. Purulent peritonitis is the form always present in perforative cases, and often without this accident is a later stage of acute peritonitis from whatever cause. In this form there are either collections of pus usually localized in small pockets formed by loops of intestine bound together by adhesions; or in a smaller number of cases mostly free in the abdominal cavity and produced in large amount. The pus has sometimes a most offensive odor, especially in perforative cases. In those dependent upon the pneumococcus it is of a greenish-yellow color and without offensive odor. In streptococcic peritonitis the secretion is yellowish or greyish and thin, and shows little tendency to encapsulation.

In all varieties of the disease the lesions may be general, or limited to certain regions as, for instance, the pelvis, the appendix, or the neighborhood of some intestinal lesion elsewhere. In localized purulent peritonitis an abscess of some size may result, walled off by inflammatory products from the rest of the peritoneal cavity. In fetal peritonitis fibrous adhesions may remain constricting the intestines or perhaps obliterating the bile-duct.

Symptoms.—The symptoms of *general* peritonitis in typical cases in childhood do not differ materially from those seen in adult life. The onset is usually sudden and severe, characterized by vomiting, high fever of 103° to 105°F. (39.4° to 40.6°C.), abdominal pain and tenderness, constipation, or very frequently diarrhea. In a short time the child presents the appearance of being very ill. The face has a pallid, strained, sunken aspect, with sharpness and coldness of the nose, hollowness about the eyes and dryness of the teeth and tongue. The expression is clearly that of pain. The pulse is rapid, small and compressible, and the breathing is shallow and rapid on account of the pain which abdominal respiration produces. The temperature continues high, but may be normal or subnormal. The abdomen is rigid, tympanitic and even meteoric. The patient lies on his back with the legs drawn up on the abdomen; the extremities are often cold and cyanotic; the mind is clear; vomiting may cease or may continue troublesome; hiccough may be present. The urine is often scanty, or there may be dysuria necessitating the employment of the catheter. The blood generally exhibits a decided leucocytosis, especially in the perforative cases, except in the very severe instances where no reaction can take place, and where even a leucopenia may be found. Later in the disease, if the course is not too short, evidences of fluid in the abdominal cavity may appear, shown by dullness in the flanks and by fluctuation. This symptom is, however, often absent.

In *localized* peritonitis the severity and the character of the symptoms depend largely upon the cause. The most frequent form is that associated with appendicitis and has already been described (p. 803). Its presence is also to be suspected whenever a child with gonorrheal vulvovaginitis suddenly develops fever with abdominal pain, tenderness and

distention. I have, however, seen this in but 2 instances. Rarely in children the inflammation is localized just below the diaphragm (*subphrenic abscess*). This is usually secondary to affections of the liver or often to pneumonia or pleurisy, and the symptoms simulate closely those of empyema. (See p. 851.)

Acute peritonitis in infancy may exhibit symptoms far from characteristic, and often so obscure that no diagnosis is made during life. Vomiting may be absent and the temperature little elevated if at all. On the other hand, it may be high, especially in the new born. The abdomen is distended and rigid; but distention of and pain in the abdomen in infancy are so common from other causes, such as colic, that the symptom is of little value. Abdominal tenderness is characteristic when present, but is often absent. In many cases, however, the symptoms are sufficiently like those of childhood to make the diagnosis clear.

Course and Prognosis.—The disease is always a serious one with uncertain outcome, the course and final issue depending largely upon the localization, the nature of the pathological lesions, and, to some extent, on the specific cause. In the most severe cases of widespread *general purulent peritonitis* the course is short and death is liable to occur in 3 or 4 days or even less. Other cases not so intense in character last a longer time, terminating fatally in 1 to 2 weeks. General peritonitis dependent upon intestinal perforation is always of a serious type and runs a rapid and fatal course; unless perhaps checked by prompt operative interference. The earlier operation is done in these cases the greater the chance of recovery. Peritonitis of the new born, like any other septic variety, is nearly always fatal. *Fibrinous* peritonitis gives a better prognosis than the purulent form and *serous* cases are still more favorable. But general peritonitis is, as a rule, a very fatal malady, with death-rate of probably 60 to 80 per cent.

In *localized* peritonitis the prognosis is much better in proportion as the area involved is small, depending, too, largely on the nature of the lesion. A small fibrinous area following upon inflammation of some neighboring region disappears with the recovery of the original lesion. There is always a danger, however, that the localized inflammation may become a general one. Purulent peritonitis may be entirely localized and may then end in recovery by discharging into the rectum, the kidneys, or through the umbilicus. The cavity remaining gradually fills with granulation tissue, and heals as does any other abscess cavity. The

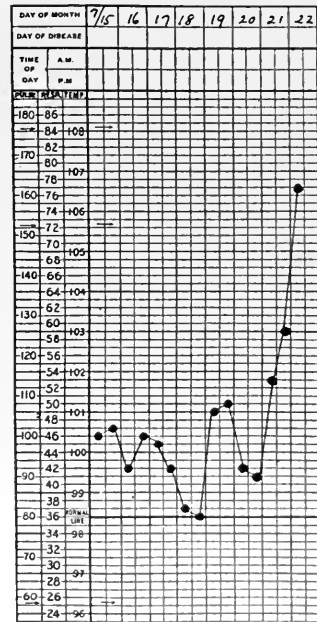


FIG. 273.—SEPTIC PERITONITIS.

John W. Entered the Children's Hospital of Philadelphia, July 15, aged 4 days. Increasing weakness, distended abdomen, moderate, irregular temperature, chiefly subfebrile until the last day. Laparotomy July 21. Post-mortem examination showed thick plastic exudate. A diplococcus, streptococcus and an undetermined bacillus were found in the fluid.

danger of extension to the general peritoneal cavity is greater in a localized purulent peritonitis than in the fibrinous form. The course is often considerably prolonged, the temperature remaining elevated, and often exhibiting a distinctly hectic type. Examination of the abdomen may reveal the presence of an abscess.

The nature of the germ exerts an influence upon the course and prognosis. *Streptococcic* peritonitis is the variety oftenest seen in sepsis in the new born (Fig. 273); after some of the infectious fevers, as scarlet fever and diphtheria; and in some of the cases of peritoneal infection from the

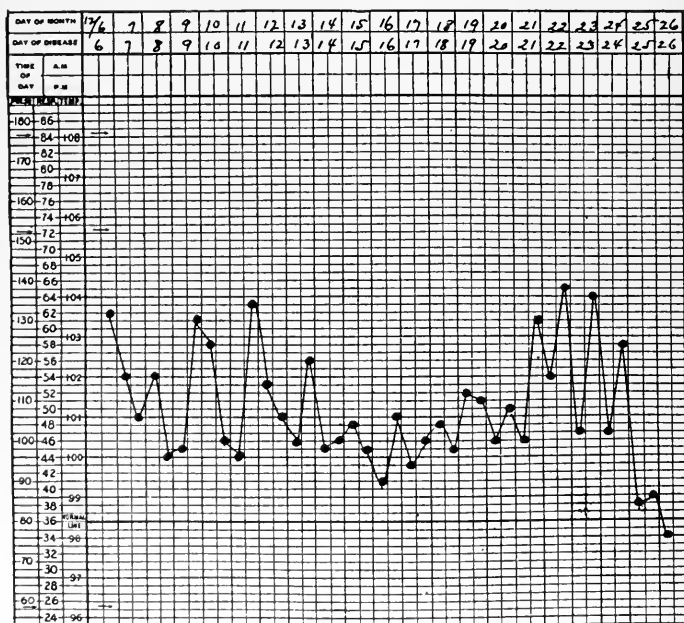


FIG. 274.—PNEUMOCOCCIC PERITONITIS; FOLLOWED BY PHLEBITIS AND EMPYEMA.

Margaret K., aged 5 years. Sudden onset Dec. 1 of apparently acute gastric indigestion, with vomiting, diarrhea and fever. Seemed convalescing in less than a week; Dec. 6, developed severe abdominal pain, fever, and diarrhea; by Dec. 17, symptoms had moderated, but physical signs of fluid in lower part of abdominal cavity had appeared. Signs of pleuropneumonia developed, and child much more ill. Abdominal condition grew worse. Operation for peritonitis done Dec. 23. 1½ quarts (1420) pus removed, containing pneumococci. Gradual improvement. Pulmonary signs cleared up. Dec. 29, phlebitis left leg, with fever; Jan. 7, empyema has developed, operated upon today; Jan. 16, convalescing. Chart shows very moderate fever even shortly before peritoneal cavity opened.

intestinal tract. The course is severe and rapid to a fatal termination. *Pneumococcic* peritonitis (Fig. 274) is oftenest preceded or attended by some affection of the lung or pleura, but may develop independently of this as a primary disorder. It often presents certain definite characteristics. After the sudden onset and early severe symptoms a remission may take place in from 6 to 8 days, but diarrhea and abdominal distention persist and the case often suggests typhoid fever. In the course of about 2 weeks fluid collects in the abdominal cavity. The general symp-

toms may remain favorable, or there may be emaciation and fever and a condition suggesting tuberculous peritonitis. The pus produced in this form shows a decided tendency to encapsulation, usually in the lower part of the abdomen or about the umbilicus, and after several weeks may be discharged in some direction, if not earlier evacuated by operative interference. In many instances, however, the inflammation finally becomes widespread; or the course may be very short from the beginning, no remission occurs, the inflammation becomes general, and the prognosis is unfavorable, the worst cases dying in a few days from the onset. The mortality in the pneumococcic cases is much less than in those dependent upon the streptococcus. Annand and Bowen¹ in 91 collected cases in children, omitting 2 in which the result was unknown, found a total mortality of 51.66 per cent. In 62 of the cases operation was performed; in 29 not. The mortality in 87 cases collected by Koos² was 39.55 per cent.

Gonococcic peritonitis develops oftenest by an extension from a vulvo-vaginitis. It may be general in nature, but usually, although the onset may be threatening, the symptoms soon abate, the inflammation is localized in the pelvic region, and, as a rule, resolution occurs. When gonococcic peritonitis is general the course, prognosis and termination are the same as in other severe forms; but this variety fortunately is rare.

Complications.—Peritonitis is oftener a sequel to other conditions than itself a primary disease. In many instances, however, there is shown a tendency for inflammation of other serous membranes to develop simultaneously with it, such as pleurisy, meningitis, or pericarditis. This is especially so in infancy, and when the disease depends upon a general septic poisoning with the streptococcus, or sometimes upon the pneumococcus.

Diagnosis.—In typical cases this presents little difficulty and rests upon the sudden onset; peculiar expression of the face; obstinate initial or continued vomiting; prostration and collapse; abdominal tenderness and distention; and later the discovery of a general or localized collection of fluid. As, however, the disease is subject to many variations the diagnosis is often difficult. *Typhoid fever* with abdominal pain and distention often resembles it closely. The matter of greatest importance is to recognize the occurrence of perforation and consequent peritonitis in this disease. This is often exceedingly difficult, especially in children. The occurrence of sudden fall of temperature followed by a rise; the sudden onset or increase of abdominal pain, tenderness, rigidity and distention; and the marked increase in the severity of the symptoms with acceleration of pulse and respiration point strongly to the development of peritonitis. I have repeatedly seen cases, however, where none of these symptoms were sufficiently characteristic to warrant a diagnosis early enough to be of service to the patient. The absence of leucocytosis may be of value in excluding peritonitis, but is not trustworthy. *Intussusception* or other obstruction of the bowel is to be borne in mind when making a diagnosis. Like peritonitis, it may cause vomiting, tympanites and constipation. The last is, however, more obstinate in intestinal obstruction, and, as intussusception is nearly always the variety of obstruction seen in children, the passage of bloody mucus with straining,

¹ *Loc. cit.*

² Archiv f. Kinderh., 1907, XLVI, 228.

and the discovery of a tumor, aid in its recognition. *Acute ileocolitis* may strongly suggest localized peritonitis due to an appendicular inflammation; and when peritonitis develops as a sequel to acute enteritis it may be with difficulty differentiated from the primary disease. *Pneumonia* and *pleurisy*, with their well-recognized tendency in some cases to develop pain referred to the abdomen, may readily simulate peritonitis. There is, however, no real abdominal tenderness in these conditions, and a careful examination of the thorax will generally serve to make the diagnosis certain. (See Appendicular Pneumonia, Vol. II, p. 80.) Pneumococcic peritonitis, when in the stage of effusion, is to be distinguished from *tuberculous peritonitis* by the more sudden and severe onset. It is to be differentiated from *appendicitis* by the absence or slight development of localized rigidity in the appendicular region. Peritonitis in infancy may exhibit typical symptoms, but when not can hardly be recognized with certainty during life.

Treatment.—The medical treatment of acute general purulent peritonitis is unsatisfactory and at the most palliative and symptomatic. The best procedure is very prompt exploratory operation to discover the cause of the inflammation and to allow a discharge of the pus from the abdominal cavity. In less severe cases, and where it is doubtful whether pus is present, and in any case where operation cannot for any reason be performed, other measures must be employed. The patient should be at absolute rest and the peristalsis and pain held in control by opiates, best given hypodermically. The early employment of free purgation by salines with the intent of favoring elimination is recommended by many, but condemned by others on the ground that it disturbs the resting of the intestines which is so greatly to be desired. It would certainly seem safer to employ frequent, large, normal saline enema to attain this end. Owing to the obstinate early vomiting it is often inadvisable to administer any food by the mouth at first, nutritive enemata and water being given by the bowel. If water is not retained hypodermoclysis may be employed to supply liquid to the system. Lavage of the stomach may be useful for the same purpose and to control vomiting. Later broth, peptonized skimmed milk, and albumen-water may be allowed in small amounts frequently repeated. The application of ice-bags to the abdomen is to be recommended, or, if this fails to relieve the pain, warm compresses or turpentine stupes may be employed. For the prostration strychnine, camphor, or adrenaline may be given hypodermically.

In cases of localized peritonitis, the need of prompt surgical aid is not always so great, depending largely upon the cause. When secondary to appendicitis, although not certainly purulent, undoubtedly the safest procedure is early operation. When the peritonitis is gonorrheal, operation is rarely indicated and the medical treatment prescribed is to be preferred. In any case of localized peritonitis where an accumulation of pus can be discovered, operation should be done, since trusting to spontaneous evacuation is an unsafe procedure. Whenever peritonitis can be determined to be pneumococcic in nature, the waiting for encapsulation or local manifestation is usually to be advised. It is always to be borne in mind that the large majority of cases of peritonitis, except in infancy, are dependent upon appendicitis and that the operative treatment as recommended for this is always to be preferred, unless it is certain that appendicitis is not the cause. (See Appendicitis, p. 800.)

SUBPHRENIC ABSCESS

This may be regarded as a localized purulent peritonitis, the peritoneum having been involved secondarily to some neighboring suppurative process. It is uncommon in children. Jopson¹ could find but 22 cases under 15 years of age in a total of 247 reported in medical literature. It may be due to pneumonia, empyema, abscess of the liver, tuberculous cavities, or appendicular abscess. The most frequent situation is above the liver; much less commonly above the spleen. The symptoms are almost identical with those of empyema, and the diagnosis is hardly possible except after operative interference, done generally with the idea that empyema is present. Occasionally the abscess contains air also, and then simulates a pyo-pneumothorax. The treatment is that for empyema.

NON-TUBERCULOUS CHRONIC PERITONITIS

The lesions and symptoms of this condition vary with the cause. A chronic, localized purulent peritonitis may follow the acute form, and it may be long before the abscess heals. In other cases there is a diffuse fibrinous inflammation, sometimes chronic from the beginning, with extensive adhesions. This may be localized especially about the spleen or the liver, or may involve the coils of the intestines, and the fibrous bands produced may result in intestinal obstruction. The disease is usually recognized only at autopsy. Fetal peritonitis has become of the chronic variety by the time of birth and numerous firm fibrous adhesions may be found post-mortem.

Chronic Ascitic Peritonitis.—This is a somewhat characteristic form of chronic peritonitis so closely resembling the tuberculous variety that many claim it is identical. Henoch² and others, however, maintain its independence. The condition is certainly an uncommon one, seen generally in later childhood, and the cause is unknown, although trauma may have an influence, and cases have been reported after measles. In this variety there is, in addition to a fibrinous inflammation with adhesions in smaller or larger degree, a large amount of serous effusion. The symptoms resemble those of tuberculous peritonitis in the gradual failure of health, loss of appetite, weakness, and abdominal distention with evidences of fluid free in the peritoneic cavity. They differ from them, however, in the absence of nodular masses discoverable on palpation, the moderate degree or even lack of abdominal tenderness, the normal or but slightly elevated temperature, and the absence of that degree of emaciation and constitutional involvement usually seen in the tuberculous variety. In fact the chief symptom is ascites for which no discoverable cause can be found. If no tuberculin reaction is obtainable, and if inoculation of guinea-pigs with the fluid procured by aspiration gives a negative result, the disease is probably not tuberculosis. The disorder is to be distinguished from simple ascites through the failure of any evidence of disease of the liver, kidneys or heart. The course and prognosis are usually favorable, the fluid being gradually absorbed. In some cases chronic peritonitis may be combined with a chronic pleurisy with serous effusion, mediastinitis, or a serous pericarditis, the condition then being in fact a polyserositis of a non-tuberculous variety.

¹ Arch. of Ped., 1901, XX1, 420.

² Vorlesung, u. Kinderk., 1895, 542.

Treatment consists in rest, the administration of diuretics and of saline purgatives in moderate amount, the maintaining of the general health by tonic measures, and eventually aspiration or even laparotomy if recovery does not take place without these.

TUBERCULOUS PERITONITIS

The great majority of the cases of chronic peritonitis are of this nature, but the disease may also develop acutely as a part of miliary tuberculosis. Respecting the frequency of its occurrence, Schmitz¹ found 24 cases in 9134 sick children examined; *i.e.* 0.26 per cent., and Cassell² 18 cases in 15,000 children; *i.e.* 0.12 per cent. It would appear to be a distinctly less common disease in this country than in Europe.

These statistics apply to tuberculous peritonitis in its clinical manifestations. From a purely pathological point of view it is a frequent form of tuberculosis, especially in early life. Thus Biedert³ in 883 collected post-mortem examinations on tuberculous children found involvement of the peritoneum to some extent in 162; *i.e.* 18.3 per cent. but many of these consisted of no more than the occurrence of scattered tubercles upon the serous membrane. In fact peritonitis of mild degree is often only a post-mortem discovery, the clinical symptoms having been those of tuberculosis elsewhere in the body.

Etiology.—In the study of cases with clinical manifestations, the influence of *age* is prominent among the predisposing causes. According to the statistics of Osler⁴ in 357 collected cases at all ages 10 were under 10 years of age; 75 from 10 to 20 years; and adults as frequently attacked as children. Many observers, however, consider the disease rather one of early life. Relative to the distribution throughout infancy and childhood, tuberculous peritonitis is uncommon in the 1st year, Weil and Pehu⁵ having been able to find only 100 cases with clinical manifestations occurring in the nursing period. Rarely, however, the disease may be even congenital (Charrin).⁶ In 306 cases reported by Faludi⁷ nearly half were between the ages of 3 and 7 years, only 8 being under 1 year; while of 161 cases studied by Fletcher⁸ 84 (52.17 per cent.) were from 1 to 5 years old.

The disease is about equally divided between the sexes (156 boys; 150 girls, Faludi). Exceptionally the development of tuberculosis of the peritoneum may appear to be incited by such causes as trauma of the abdomen; or the occurrence of some infectious disease may exert an influence, as with other forms of tuberculosis. In the great majority of cases no such cause can be discovered. In these the process may develop apparently simultaneously in the peritoneum and elsewhere in the body, as in the pleura; or may be secondary to lesions in the lungs, the intestine, or the mesenteric or other lymphatic glands, or bones; or be one of the evidences of a general miliary tuberculosis. Oftenest, from a purely clinical standpoint, tuberculous peritonitis seems to be the primary or sole manifestation of tuberculosis, and probably in some instances it is in fact so; but certainly much more frequently it is sec-

¹ *Jahrb. f. Kinderheilk.*, 1897, XLIV, 316.

² *Deut. med. Wochenschr.*, 1900, XXVI, 596.

³ *Jahrb. f. Kinderheilk.*, 1884, XXI, 158.

⁴ *Johns Hopkins Hosp. Rep.*, 1891, II, 67.

⁵ *Archiv de méd. des enf.*, 1909, XII, 415.

⁶ *Lyon méd.*, 1873, XIII, 295.

⁷ *Jahrb. f. Kinderheilk.*, 1905, LXII, 304.

⁸ Garrod, Batten and Thursfield, *Diseases of Children*, 1913, 242.

ondary to some small or perhaps undiscovered remote tuberculous lesion. The question of the type of tubercle bacillus oftenest present in the different manifestations of tuberculosis has been considered elsewhere. (See p. 542.) Tuberculous peritonitis is probably one of the most frequent varieties dependent upon the bovine bacillus. Concerning the method of invasion, tubercle bacilli may reach the peritoneum either through the general circulation, as in cases of widespread acute tuberculosis; from the intestinal tract by way of the lymphatic vessels and the mesenteric glands, with or without a discoverable primary intestinal lesion; or, still oftener, from other regions of the body. Indeed the combination of tuberculous peritonitis with intestinal tuberculosis is often absent. In Borschke's¹ 226 cases of tuberculous peritonitis 86 had the intestine intact.

Pathological Anatomy.—Examination made after laparotomy or autopsy in fatal cases of acute miliary tuberculosis shows a dissemination of grey miliary tubercles over the involved portion of the peritoneum. These tubercles may disappear, if the case recovers, or may increase in size and become more or less confluent into larger masses, which may show caseous degeneration in the central parts. The serous membrane is congested, thickened, and exhibits more or less fibrinous exudation, while a certain amount of serous, seropurulent, or even hemorrhagic effusion is always present, although the last is infrequent in children. The omentum is usually much involved and may be greatly thickened with large tuberculous nodules, or shrunk, or rolled up into a firm mass. The relative amount of the fibrinous and of the fluid exudate varies with the subject. When the former predominates and there is very little fluid the coils of the intestines are firmly bound together and to the abdominal wall and the various viscera by dense adhesions. In these cases the condition is analogous to that seen in fibroid phthisis. In other instances the fluid is in large amount, either free in the peritoneal cavity or encapsulated when of longer duration, and there is only a moderate amount of fibrin on the serous membrane, and some scattered and easily torn adhesions are present. Many cases show the development of both conditions in various degrees, and in either form the process may go on to recovery, more or less trace of peritonitic inflammation perhaps remaining permanently, with encapsulation of any caseous masses which have been produced. In still other cases the large tuberculous caseous masses which have developed, especially in the fibrous form, finally produce pus, which discharges itself through the abdominal wall especially at the navel, or by ulceration into the intestine.

Symptoms.—Based upon the complex of symptoms and upon the anatomical lesions various classifications of tuberculous peritonitis have been made. Neither symptoms nor lesions, however, allow of any entirely sharp division, since intermediate forms occur. The following may be utilized as a method of convenience in study: (1) *The ascitic form.* (2) *The fibrous, adhesive, or plastic form.* (3) *The caseous, or ulcerative form.*

These varieties are all more or less chronic and, as stated, shade into each other. In addition there is the development of miliary tubercles upon the peritoneum attending cases of acute general miliary tuberculosis, but producing no certain clinical manifestations, and not considered here. In some such cases, it is true, the abdominal symptoms predominate, and there are abdominal pain and tenderness, with rapid develop-

¹ Virchow's Archiv, 1892, CXXVIII, 121.

ment of effusion, while the other symptoms are the severe constitutional manifestations of acute miliary tuberculosis (p. 547). Attention will be given in what follows to the chronic forms only.

1. The Ascitic Form.—The development of symptoms may be entirely insidious, or may be marked by gradually increasing loss of health, flesh and strength, vague or slight abdominal pain and tenderness, and irregular fever of moderate degree. There may be vomiting, but this is not a characteristic symptom, and the bowels may be constipated or unaffected, or, not infrequently, diarrheal. There is nothing on which to base a diagnosis until finally attention is called to the gradually increasing distention of the abdomen, which may, indeed, be the first symptom of any sort noted. This is at first tympanitic, but later exhibits fluid free in the abdominal cavity, and giving the ordinary signs of fluctuating dullness in the flanks, shifting with change of position, and with upward displacement of the diaphragm and of the hepatic dullness, flattening or pouting of the umbilicus, and dilatation of the abdominal veins. When the fluid is in small amount careful examination may be required to show its presence, and the distention is principally tympanitic. Sometimes the thickened omentum or enlarged lymphatic glands can be detected. The fluid is usually chiefly serous, and straw-colored or slightly brownish.

In exceptional cases the inflammation may begin much more suddenly and severely, and may strongly suggest appendicitis if the pain and other evidences of the tuberculous disease are at all marked in the appendicular region.

2. The Fibrous Form.—This is of more frequent occurrence than the ascitic variety. The early symptoms are very similar to those described, but the onset is even more gradual and the development slower. Fever is still less prominent and is often absent (Fig. 275). The general health is at first little affected. Enlargement of the abdomen finally attracts attention, but this is usually found to be tympanitic in nature. Careful palpation may reveal nothing abnormal, or there may sometimes be found nodules, or areas of dullness on percussion which are not limited to the flanks and which do not alter with change of position of the patient. These signs depend upon the development of tuberculous deposits in the abdominal wall, in the intestines bound to it, in the omentum, or in the mesenteric glands, or sometimes are produced by fluid confined to a certain region by adhesions. In some cases there results a distinct alteration of the shape of the abdomen as determined by inspection. This may be globular, as in the ascitic form, or irregular in outline dependent upon the presence of adhesions. There is usually little or no fluid present, but sometimes it may be in large amount. It may be serous or seropurulent. Abdominal pain and tenderness are absent or slight. The formation of tuberculous masses or of firm contracting adhesions may proceed so far that secondary symptoms result, due to pressure upon various regions. Among these are edema from interference with the circulation; digestive disturbance from pressure upon the intestine or stomach; intestinal obstruction; and abnormal urinary conditions. Fibrous peritonitis may be such from the beginning, or may be a sequel to the ascitic form first described. The chief characteristic is the development of the fibrous exudate, and the presence of many large nodules suggests a combination with the caseous type.

3. The Caseous or Ulcerative Form.—This severe type of tuberculous peritonitis may be a sequel of the others described, or may less commonly

exhibit its peculiarities from the beginning. Its chief characteristic is the formation of large tuberculous masses and smaller nodules which undergo caseation and often produce pus. Owing to this and to the fact that a tuberculosis in other parts of the body is generally present also, the symptoms early become severe. There is more constant and greater elevation of temperature, which often assumes a hectic type (Fig. 276);

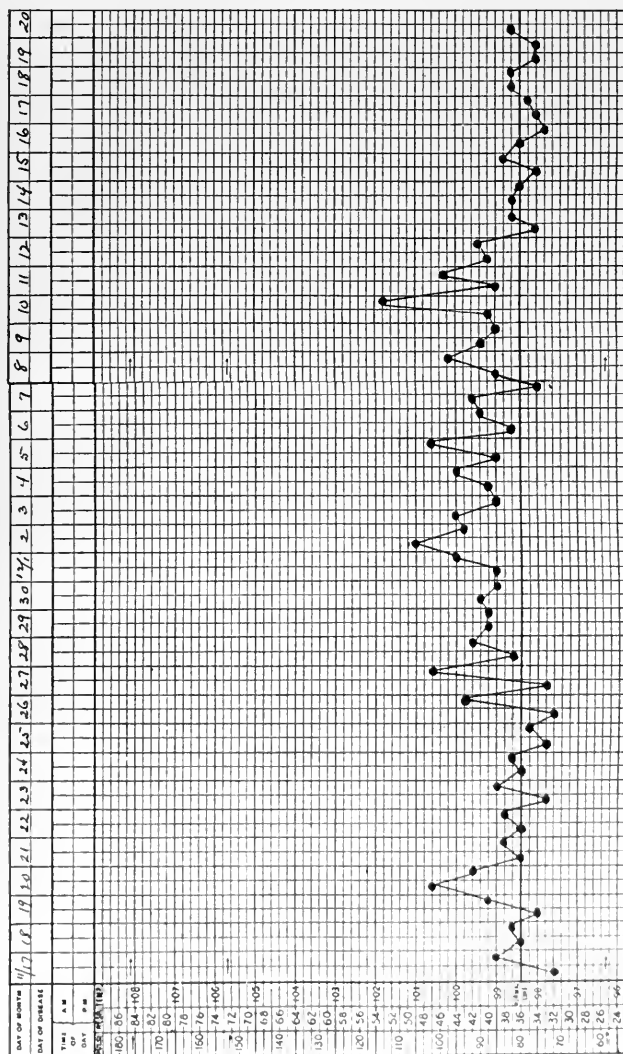


FIG. 275. TUBERCULOUS PERITONITIS, FIBROUS FORM. ALMOST WITHOUT FEVER.

Robert V., aged 2 years, a patient in the Children's Hospital of Philadelphia. Symptoms appeared in April. Large abdomen and loss of appetite. Mass could be felt through abdominal walls. Laparotomy done Dec. 1, showed adhesive type of tuberculous peritonitis. Discharged improved.

diarrhea is frequent; there are abdominal pain and tenderness; and emaciation, anemia, and loss of strength are progressive and decided. The abdomen becomes distended, but, although fluid which is often purulent may be present in considerable quantities, the matting of the intestines and the presence of the large tuberculous masses frequently prevents the discovery of this by percussion or by palpation. The abdomen may give on palpation a very characteristic sensation of doughy resistance.

Pus may make its way toward the umbilicus or elsewhere, the evidences of beginning abscess of the abdominal wall being clearly visible.

Course and Prognosis.—These vary greatly with the nature of the lesion. In the *ascitic* form without marked fibrous change, the least severe variety, the course is from 1 to several months. In favorable cases the effusion varies but little in amount from time to time, or may exhibit periods of temporary diminution or increase; but finally it gradually disappears completely, while fever lessens, the general health improves, and recovery takes place. Adhesions may, however, remain, or relapse may later occur, or tuberculosis make its appearance in other parts of the body. In the more unfavorable cases emaciation increases,

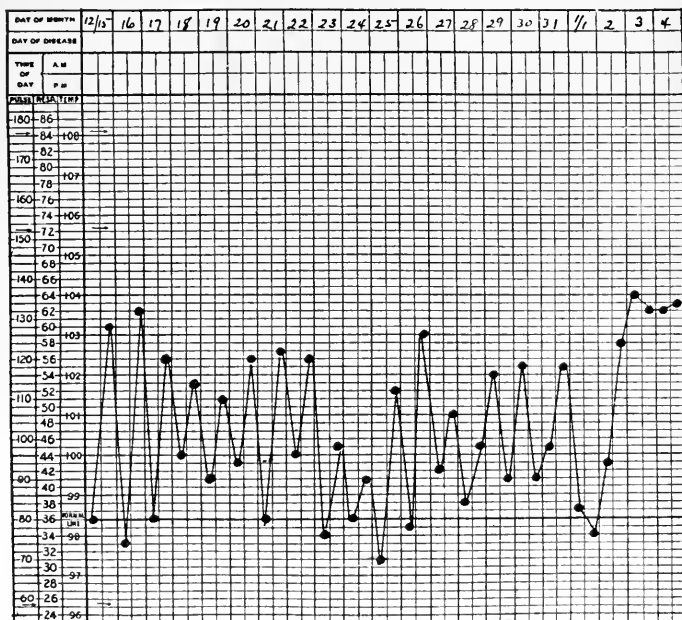


FIG. 276.—TUBERCULOUS PERITONITIS, CASEOUS FORM, WITH HECTIC TYPE OF TEMPERATURE.

Elizabeth R., aged 11 years. Said to have been taken ill June, 1914. Admitted to the Children's Hospital of Philadelphia, Dec. 15, 1914. Operation Dec. 29, showed fibro-caseous tuberculosis of the peritoneum. Died March, 1915. Principal symptoms progressive loss of health, with fever.

and death takes place from exhaustion or following tuberculosis of some other organ; or the case passes into the fibrous or the caseous type.

In the *fibrous* form the course is slower, lasting several months or even a year or more. It may then slowly undergo recovery, but leaves adhesions remaining, even after apparent cure; and there is a decided tendency to relapse. This variety may prove fatal by gradually exhausting the patient; or it may pass into the caseous form and kill through the influence of pus-production; or may be followed by the development of tuberculosis elsewhere in the body.

The *caseous* form is of shorter duration than the two just described, lasting usually only a few months after evidences of the breaking down of the lesions show themselves. The duration is influenced considerably by the course of the tuberculosis usually present in other parts of the body.

The prognosis of tuberculous peritonitis in general is determined by various circumstances, such as the inherent strength and resisting power of the patient; the type of the disease; the age of the child; and the presence or absence of tuberculosis in other regions. The younger the patient, the worse is the prognosis. In Fletcher's¹ 163 cases with 49 deaths, 35 were in children less than 4 years of age. Of course, the presence of tuberculosis elsewhere greatly adds to the liability of a fatal termination. The prognosis of the ascitic form is not as unfavorable as formerly believed, now that the treatment of the malady is better understood. It is in this type especially that laparotomy has often proven of value; but that it accomplishes all that was at first claimed for it is doubtful, and it is still a question whether the results obtained by it are superior to those with non-operative measures. The fibrous type gives a more unfavorable prognosis, and certainly the larger number do not recover; not so much from the existence of this form itself, as because of the tendency to pass into the caseous type, or to give rise to serious complications. Laparotomy may do good here also, but less is to be expected than in the ascitic cases. The caseous type with severe constitutional symptoms gives a most unfavorable prognosis, but is yet capable of cure, if other parts of the body are not involved.

Regarding the statistical prognosis in general and the influence of treatment, statements vary to a considerable extent; and are, moreover, at fault, because it is usually not known in how many instances a fatal relapse or a development of tuberculosis elsewhere finally occurred. In general the proportion of final complete recoveries might be placed at approximately 30 to 50 per cent. As regards the value of *operative* treatment, one of the earliest and most favorable series is that of Aldibert,² of 52 children operated upon with 7 deaths, a mortality of 13.46 per cent. Faludi³ reports on 70 cases of which 46 were operated upon and 24 not. Of the former 45.5 per cent. recovered, and of the latter 20.83 per cent. Many of the cases were watched for several years after operation. Schramm⁴ in 45 cases found permanent recoveries, in those which could be traced, in 20 per cent. of those treated medically and 75.4 per cent. of the operated cases. Kissel⁵ reported upon 54 cases, 35 of which were operated upon, with 27 recoveries; *i.e.* 77.14 per cent.

In favor of *medical* treatment is the report by Sutherland⁶ of 41 cases, 27 of which were treated medically, with recoveries in 22; *i.e.* 81.3 per cent and 14 surgically, with 7 recoveries; *i.e.* 50 per cent. Most of the cases were under observation for over a year. Borchgre-vink⁷ in 22 cases treated medically had 19 permanent recoveries; *i.e.* 81.82 per cent.

Complications.—These are principally the occurrence of manifestations of tuberculosis elsewhere in the body. Tuberculous pleurisy is not infrequently associated, as is less often tuberculous pericarditis, and tuberculosis of the meninges or of the lungs is a not uncommon final cause of death. The genital organs are only occasionally secondarily attacked in children. Involvement of the intestines and mesenteric glands is frequent. To be noted also are septic processes from a collec-

¹ *Loc. cit.*, 250.

² Thèse de Paris, 1892.

³ *Loc. cit.*

⁴ Wiener med. Wochenschr., 1903, LIII, 354.

⁵ Arch. f. klin. Chir., 1902, LXV, 373.

⁶ Arch. of Ped., 1903, XX, 81.

⁷ Mittheil. aus der Grenzgeb. d. Med. and Chir., 1900, VI, 434.

tion of pus, or the rupture of an accumulation of this; amyloid changes in the liver and spleen; and intestinal obstruction or other pressure-symptoms of various sorts produced by the fibrous adhesions.

Diagnosis.—This is by no means easy except in typical and well-advanced cases. Early in the attack it is usually impossible. It rests principally upon slow failure of health; irregular fever without discoverable cause; slight abdominal pain and tenderness; and finally decided abdominal enlargement with the discovery of fluid or of evident thickening of the abdominal wall and the presence of masses beneath it. The existence of tuberculosis elsewhere in the body is suggestive. The employment of the cutaneous tuberculin test is of value if negative, but a positive reaction does not determine that the abdominal condition is tuberculous. Moreover, severe cases may have lost the power to respond to the von Pirquet reaction. The examination of the blood is of some value, there being in tuberculous peritonitis an absence of the increase of leucocytes characteristic of most inflammatory conditions. Ascites of tuberculous nature is to be distinguished from that due to other causes. That dependent upon *cardiac* or *renal disease* is accompanied by dropsy elsewhere, while other characteristic symptoms are present as well. *Atrophic cirrhosis* of the liver gives diminution in the size of this organ, and icterus may attend. In doubtful cases examination of the fluid obtained by puncture will probably show in tuberculosis an increase in the number of lymphocytes, and inoculation experiments will produce the disease in animals (Durante).¹ Puncture is, however, a procedure not without danger of perforation of the intestine. Tubercle bacilli are not often discovered microscopically. *Chronic non-tuberculous peritonitis* is distinguishable by the absence of discoverable nodular masses, and of so decided a degree of emaciation and loss of health. The presumption should always be that chronic peritonitis is tuberculous unless certainly proven otherwise. Chronic *enteritis* with diarrhea, abdominal distention, and wasting may resemble tuberculous peritonitis, but the history of the case and continued observation will generally remove any doubt.

Tuberculous peritonitis with masses discovered through the abdominal wall is to be distinguished from *abdominal tumors* of other nature, as well as from fecal accumulations in the colon. The existence of other symptoms characteristic of tuberculosis is of service here. That the disease is of the caseous type is rendered probable, when, in addition to the presence of the tuberculous nodules discovered by palpation, there are decided abdominal pain and tenderness, tympanitic distention, and severe constitutional symptoms. The existence of tuberculosis in other regions aids in the diagnosis.

Treatment.—The medical treatment consists in the employment of all measures which will aid the patient to overcome a tuberculous process of any nature. (See p. 562.) Rest lying down should be enforced, but this by no means signifies confinement to bed in the house. On the other hand, the patient should be kept in the open air as much as possible and preferably removed to some climate which favors this. Sojourn at the mountains and especially at the seashore has been of great service in many instances. The diet should be digestible and abundant, and there is no reason to abstain from a highly nitrogenous regimen because fever is present. On the contrary, the food should be stimulating. The presence of diarrhea and other digestive disturbances often renders

¹ La Pediatría, 1901, IX, 437.

satisfactory feeding difficult; yet sufficient nourishment is one of the most important therapeutic factors. Alcoholic stimulants may be given as required. Local application of mercurial ointment, of ichthyol, and of iodoform have been recommended, as has been the exposure of the abdominal walls to the direct rays of the sun. The internal administration of creosote and of iodoform have each their adherents. Tonic remedies may be needed, including cod-liver oil if it is well tolerated, and such treatment also as complications demand. In general, however, the chief dependence is to be placed upon hygiene and diet. What benefit is to be gained by the injection of tuberculin is still an unsettled question. There is some reason to believe that it is of more value here than in some other forms of tuberculosis, but further experience is demanded.

Operative interference in the form of laparotomy is to be seriously considered in the light of the data given regarding it. Certainly it seems to be of great possible value if done early in the ascitic cases, and since the danger of operation is slight and that of the disease itself great, and as recovery from the disease is often very prompt after it, it should be recommended in all cases of this type, if a few weeks of thorough hygienic and dietetic treatment have been without influence. In the purely fibrous form without tuberculous masses recovery may take place spontaneously; but even here laparotomy may do much good and can do little harm. Cases where a small localized or more generalized accumulation of pus can be discovered require operation.

TUMORS OF THE PERITONEUM

These are of occasional occurrence in early life and may be either malignant or benign in nature; primary or secondary. Cysts of various sorts are sometimes found including dermoid, hydatid, chylous and serous: They are oftenest in the mesentery. Carcinoma is very rare even when secondary. Sarcoma, fibroma, angioma and lipoma may occur. The last has sometimes attained a large size.

ASCITES

By this title is designated an accumulation of fluid in the general peritoneal cavity. The condition is not a disease, but a symptom dependent upon various causes. It is, however, sufficiently common and important to warrant separate brief consideration. The fluid may be entirely clear and either colorless or of a yellowish tint; or it may be more or less hemorrhagic or contain flakes of lymph and other evidences of inflammation. Microscopically it may show either few cellular elements when it is of the nature of a transudate, or many when the cause is a local inflammatory condition. If the inflammation is a tuberculous peritonitis, the lymphocytes are in excess; if an acute peritonitis dependent upon other germs, the polymorphonuclear cells predominate.

Among the causes may be mentioned peritonitis of various sorts, as the tuberculous and the non-tuberculous forms, including polyserositis; cirrhosis of the liver, a rare cause in early life; obstruction to the portal circulation from other sources, such as pressure of a tumor or of an enlarged lymphatic gland; great anemia; and renal or cardiac disease. These last two are the most common causes. It may also develop without discoverable agency and be associated with hydrothorax and anasarca, as in the essential edema of children.

Ascites may in rare cases be congenital, is uncommon in infancy, somewhat less so in early childhood, and begins to be observed oftener in later childhood. The symptoms are similar to those seen in adult life. The abdomen is distended; there is often flattening or pouting of the umbilicus; fluctuation on palpation, with a distinct wave-like impulse obtained by sharp tapping with the hand; and dullness on percussion which shifts with change of position unless the amount of fluid is very large.

The course, prognosis and treatment depend entirely upon the cause.

Chylous Ascites.—This is an uncommon form of ascites at any time of life. The causes are variable. In the instances of true *chylous* effusion there has been an injury to, or pressure upon, the thoracic duct or some of its tributaries, as a result of which the chyle has entered the abdominal cavity. In these cases the fluid has the appearance of milk and contains fat-cells in very large numbers. In other cases the fluid is *chyliform* only, and the condition depends upon tuberculous or non-tuberculous chronic peritonitis, and the color appears to be the result of degeneration of the inflammatory products, since cells showing evidences of this are discoverable. Chylous ascites has also been produced by the rupture of a mesenteric cyst. The prognosis is, as a rule, unfavorable; yet recovery after operative interference has been reported (Huber and Silver).¹

¹ Amer. Journ. Dis. Child., 1914, VIII, 50.

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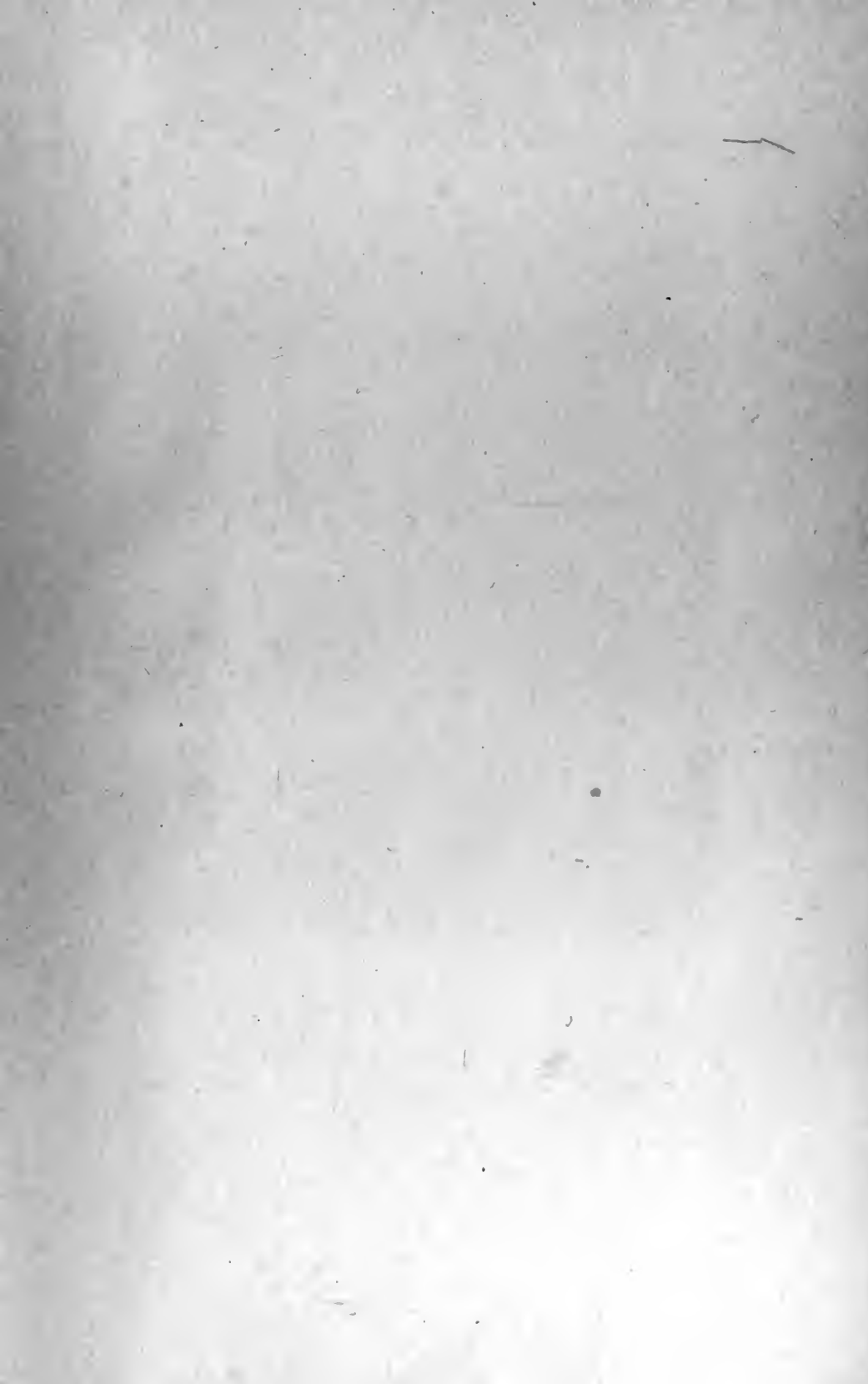
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